Digitized by the Internet Archive
in 2007 with funding from
Microsoft Corporation

http://www.archive.org/details/manualoftoxicolo00reesiala

MANUAL OF TOXICOLOGY.

A MANUAL OF TOXICOLOGY,

INCLUDING THE CONSIDERATION OF THE

NATURE, PROPERTIES, EFFECTS, AND MEANS
OF DETECTION

OF

POISONS,

MORE ESPECIALLY IN THEIR MEDICO-LEGAL RELATIONS.

BY

JOHN J. REESE, M.D.,

PROFESSOR OF MEDICAL JURISPRUDENCE AND TOXICOLOGY IN THE UNIVERSITY OF PENNSYLVANIA;
PHYSICIAN TO ST. JOSEPH'S HOSPITAL; MEMBER OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; HONORARY MEMBER OF THE NEW YORK MEDICO-LEGAL SOCIETY.

PHILADELPHIA:
J. B. LIPPINCOTT & CO.
1874.

Entered, according to Act of Congress, in the year 1874, by
J. B. LIPPINCOTT & CO.,
In the Office of the Librarian of Congress at Washington.

TO

GEORGE B. WOOD, M.D., LL.D.,

EMERITUS PROFESSOR OF THE THEORY AND PRACTICE OF MEDICINE IN THE UNIVERSITY OF
PENNSYLVANIA; PRESIDENT OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA;
PRESIDENT OF THE AMERICAN PHILOSOPHICAL SOCIETY, ETC.,

THIS WORK

IS DEDICATED,

AS A TRIBUTE OF ESTEEM FOR HIM AS A PUBLIC TEACHER,

AND OF

SINCERE PERSONAL REGARD AS A PRECEPTOR,

BY HIS FRIEND AND FORMER PUPIL,

THE AUTHOR.

PREFACE.

A NEW work on Toxicology will doubtless be regarded by some as uncalled for, since there already exist so many excellent treatises on this subject, both in our own and in foreign languages. This objection, however, might be equally urged against new publications in almost every department of science: there is scarcely one of which it may not be affirmed that its literature is already abundantly supplied. Yet this does not deter new authors from venturing before the public, prompted, doubtless, by the desire of offering something of at least a passing value, and of adding, it may be, a fragment to the store of human knowledge.

The author is fully aware that the field of investigation on which he has entered has already been very thoroughly explored by others. Names illustrious in the annals of medical and toxicological science are identified with this subject throughout the civilized world. Yet he ventures to hope that the present treatise will be found a useful text-book, both by the student and the teacher of Toxicology. From an experience of a number of years, he may presume to speak with some authority to the former, as likewise with some degree of confidence to the latter, upon topics with which he is personally familiar. His aim has been to convey his meaning in the simplest style and phraseology, believing this to be the most efficient method of teaching others.

The recorded experience of authorities becomes the common property of all who may receive it; and it may be freely used by them, and reproduced, provided always that due credit is awarded to the originals. Influenced by this view, the author feels that he needs no apology for his free quotations from standard toxicological and chemical authorities, both native and foreign. Among the former, he would specially mention Wharton and Stillé's "Medical Jurisprudence," the last edition (1873) of which is so copious and complete; and Prof. Wormley's beautifully illustrated "Micro-Chemistry of Poisons." To the latter he is particularly indebted for hints in testing for very minute portions of certain poisons. Among foreign authorities, the classic treatises of Professors Taylor and Guy, of London, of Sir R. Christison, of Edinburgh, of Orfila and Tardieu, of Paris, and of Professor Casper, of Berlin, deserve especial notice: each has contributed not a little to the completeness of the present volume, as the reader will perceive by the numerous references to their names in the following pages.

More than a passing reference is due to Dr. A. S. Taylor, of London, from whose elaborate works, "On Poisons," and "Principles and Practice of Medical Jurisprudence," 1873, the author has enriched his present treatise, especially in the matter of illustrative cases, as also in some other particulars.

It will be observed that more particular attention, in the details, has been bestowed upon certain special poisons, as, for example, Arsenic, Phosphorus, Opium, Strychnia, etc., because of their relatively greater importance to the toxicologist. In the first portion of the work, a chapter has been devoted to the subject of "Post-mortem Imbibition of Poisons," and another to the "Duties and Privileges of Medical Experts,"—topics which the author believes have not received sufficient consideration, and which possess very great importance for the toxicologist and the legal physician.

The subject of *spectrum-analysis* has not been treated of in the present volume. This truly beautiful method of analytical research has developed the most wonderful results both in chemistry and in other departments of science. In point of delicacy, it far transcends the most subtle and refined chemical reactions; and as a corroborative means of evidence, it will doubtless prove of great value to the toxicologist. But as it deals, so to speak, with infinitesimals, we do not think that it would be safe, in a case of alleged poisoning, to rest the evidence solely upon the spectral demonstration of the supposed toxic agent, to the exclusion of the recognized *chemical* tests. When an accumulated experience with spectral analysis has rendered the identification of the various poisons absolutely and *exclusively* certain, we can probably afford to abandon altogether the more tedious and complex methods of chemical research.

The author commits his work to the profession with the assurance that he has striven to perform his part carefully and conscientiously, and promises to profit by any friendly criticisms, if his book should have the good fortune to reach another edition.

PHILADELPHIA, 1840 GREEN STREET,
March, 1874.

CONTENTS.

CHAPTER I.

PAGE
Preliminary Considerations. — Definition of a Poison. — Deleterious substances not properly poisons 13

CHAPTER II.

Mode of action of Poisons on the animal economy.—Local and remote effects of poisons 18
Section I. The different modes by which Poisons gain access to the various organs of the body.—Nervous communication, or Sympathy.—Contiguity of structure 20
Section II. Absorption of Poisons.—Circumstances influencing Absorption.—Subsequent disposition of the poison.—Elimination of poisons.—Cause of death by poisons 22

CHAPTER III.

Circumstances which modify the action of poisons. — Quantity and Form.—Modifying influence of the part of the body to which the poison is applied.—Influence of Habit—of Idiosyncrasy—of Disease—of Tolerance 33

CHAPTER IV.

Post-mortem Imbibition of Poisons. — Its influence in medico-legal cases 38

CHAPTER V.

Evidences of Poisoning 45
Section I. Evidences afforded by the Symptoms. — Diseases resembling Poisoning 45
Section II. Evidences derived from Post-mortem appearances.—Lesions common to Disease and Poisoning 56
Section III. Inspection of the body.—Collection and preservation of the suspected materials 62
Section IV. Evidence from Chemical Analysis.—Causes of failure in the Chemical Analysis —Objects of the Chemical Analysis. — Precautions to be observed. — Accuracy of the Analysis.—Impurities in Reagents 67

PAGE

SECTION V. Evidence derived from experiments on animals.—Precautions to be observed 80

SECTION VI. Evidence derived from the circumstances (Moral Evidence) 87

CHAPTER VI.

Compound Poisoning.—Antagonism of Poisons.—Case of Dr. Paul Schoeppe 91

CHAPTER VII.

Method of Chemical Procedure in a case of suspected Poisoning.—Dialysis 98

CHAPTER VIII.

Medico-legal questions connected with Poisoning 114

CHAPTER IX.

Duties and Privileges of Medical Experts.—Subpœnas.—Compensation.—Defrauding of Experts 119

CHAPTER X.

Classification of Poisons 132

CHAPTER XI.

CLASS I. Irritant Poisons 135

SECTION I. Poisoning by Sulphuric Acid.—Symptoms.—Fatal period.—Fatal quantity.—Treatment.—Post-mortem appearances.—Absorption and elimination.—Chemical analysis.—Detection in organic matters.—Detection of spots.—Quantitative analysis 137

SECTION II. Poisoning by Nitric Acid.—Symptoms.—Fatal dose.—Fatal period.—Treatment.—Post-mortem appearances.—Chemical analysis.—Detection in organic matters.—Absorption and elimination.—Examination of suspected stains . . 149

SECTION III. Poisoning by Hydrochloric (Muriatic) Acid.—Symptoms.—Fatal dose.—Post-mortem signs.—Detection in organic matters.—Examination of suspected stains.—Quantitative analysis 158

SECTION IV. Poisoning by Oxalic Acid.—Symptoms.—Fatal dose.—Fatal period.—Treatment.—Morbid appearances.—Chemical analysis.—Detection in organic mixtures.—Quantitative analysis.—Examination of stains.—Poisoning by Binoxalate of Potassa (Salt of Sorrel) 162

SECTION V. Poisoning by Tartaric Acid—Citric Acid—Acetic Acid 173

CHAPTER XII.

PAGE
Poisoning by the Alkalies and their Salts; also by the Earthy Salts . 175

SECTION I. Poisoning by Potassa, Soda, and Ammonia.—Symptoms.—Fatal dose.—Treatment.—Morbid appearances.—Chemical properties.—Chemical analysis of Potassa and its Carbonate.—Fallacy.—Chemical analysis of Soda and its Carbonate.—Chemical analysis of Ammonia and its Salts.—Detection in organic mixtures 175

SECTION II. Poisoning by the Alkaline and Earthy Salts.—Nitrate of Potassa.—Bitartrate of Potassa.—Sulphate of Potassa.—Alum.—Chlorinated Potassa and Soda.—Salts of Baryta . 184

CHAPTER XIII.

Irritants having remote specific properties 193

SECTION I. Poisoning by Phosphorus.—Symptoms.—Chronic Poisoning.—Fatal dose.—Fatal period.—Treatment.—Morbid Appearances.—Diagnosis.—Chemical analysis.—Process of Mitscherlich and Lipowitz.—The Hydrogen method.—Phosphoric Acid.—Tests.—Amorphous or Allotropic Phosphorus . 194

SECTION II. Poisoning by Iodine and Bromine.—Symptoms.—Chemical analysis 209

CHAPTER XIV.

Poisoning by Arsenic.—Metallic Arsenic.—Arsenious Acid.—Properties.—Symptoms.—Chronic poisoning.—First appearance of symptoms.—External application.—Fatal dose.—Fatal period.—Post-mortem appearances.—Antiseptic power.—Treatment.—Chemical analysis.—Reduction process.—Liquid Tests.—Sulphuretted Hydrogen Test.—Marsh's Test and modifications.—Reinsch's Test.—Fallacies.—Bloxam's method.—Detection in organic mixtures.—Separation from the tissues.—Other preparations of Arsenic 214

CHAPTER XV.

Poisoning by Antimony.—Tartar Emetic.—Properties.—Symptoms.—Fatal dose.—Fatal period.—Post-mortem appearances.—Treatment.—Slow poisoning.—External application.—Chemical analysis.—Sulphuretted Hydrogen Test.—Galvanic Test.—Marsh's Test.—Detection in organic mixtures.—Importance of obtaining the metal.—Recovery from the tissues.—Chloride of Antimony 253

CHAPTER XVI.

Poisoning by Mercury.—Corrosive Sublimate.—Properties.—Symptoms.—External application.—Fatal dose.—Fatal period.—

PAGE

Mortality.—Treatment.—Post-mortem appearances.—Chronic poisoning.—Chemical analysis.—Reinsch's Test.—Galvanic Test.—Detection in organic mixtures—in the tissues and urine.—Relation to salivation.—Quantitative determination.—Other preparations of Mercury 272

CHAPTER XVII.

Poisoning by Copper.—Accidental poisoning.—Salts of Copper.—Symptoms.—Fatal dose.—Fatal period.—Treatment.—Morbid appearances.—Chemical analysis.—Detection in organic mixtures—in the stomach and its contents—in the tissues—in the urine.—Quantitative estimate 289

CHAPTER XVIII.

Poisoning by Lead.—Accidental poisoning.—External application.—Contamination of drinking-water.—Acetate, or Sugar of Lead.—Symptoms.—Fatal dose.—Fatal period.—Treatment.—Post-mortem appearances.—Chronic poisoning.—Painter's colic.—Lead-palsy.—Chemical analysis.—Detection in organic matters—in the stomach—in the tissues—in the urine.—Quantitative determination 305

CHAPTER XIX.

Poisoning by Zinc, Bismuth, Tin, Iron, and Chromium . . . 322

Section I. Poisoning by Zinc.—Sulphate of Zinc.—Symptoms.—Morbid lesions.—Chloride of Zinc.—Treatment.—Tests.—Detection in organic mixtures 322

Section II. Poisoning by Bismuth.—Subnitrate of Bismuth.—Contamination with arsenic.—Tests 327

Section III. Poisoning by Salts of Tin.—Preparations of Iron.—Preparations of Chromium.—Bichromate of Potassa.—Chemical analysis 329

CHAPTER XX.

Vegetable and Animal Irritants 331

Section I. Vegetable Irritants.—Drastics.—Croton Oil.—Elaterium.—Aloes.—Colocynth.—Colchicum.—Castor-oil seeds.—Savin 331

Section II. Poisoning by Black, Green, and White Hellebore.—Veratria 337

Section III. Poisoning by Carbolic Acid.—Symptoms.—Fatal quantity.—Chemical analysis.—Poisoning by Yellow Jessamine 342

Section IV. Poisonous Mushrooms (Fungi)—Symptoms . . 345

CHAPTER XXI.

PAGE

Animal Irritants 349

SECTION I. Poisoning by Cantharides.—Symptoms.—Fatal quantity.—Treatment.—Post-mortem signs.—Cantharidin . . . 349

SECTION II. Poisonous Animal Food.—Sausage-Poison.—Trichiniasis.—Cheese-Poison 353

SECTION III. Poisonous Fish.—Mussels 356

SECTION IV. Poisoning by Putrescent Food.—Poisoned Meat . 358

CHAPTER XXII.

CLASS II. Neurotic Poisons.—ORDER I.—Cerebral Neurotics . . 361

SECTION I. Narcotics.—Poisoning by Opium.—Symptoms.—Fatal period.—Fatal dose.—External application.—Morbid appearances.—Treatment.—Morphia.—Fatal dose.—Chemical analysis of Opium.—Detection of Morphia.—Meconic Acid.—Tests.—Narcotina.—Codeia.—Narceine.—Thebaia.—Meconin.—Detection of Opium in organic mixtures.—Examination for Morphia alone.—Detection in the tissues and blood . . 361

SECTION II. Poisoning by Alcohol.—Post-mortem appearances.—Diagnosis.—Chemical analysis.—Detection in the tissues . 385

CHAPTER XXIII.

Anæsthetics 389

SECTION I. Poisoning by Ether.—Symptoms.—Morbid lesions.—Chemical analysis 389

SECTION II. Poisoning by Chloroform.—Effects and symptoms.—Post-mortem appearances.—Treatment.—Analysis.—Hydrate of Chloral.—Chemical properties.—Detection in the body . 390

CHAPTER XXIV.

ORDER II.—Spinal Neurotics, or Tetanics.—Poisoning by Nux Vomica.—Strychnia.—Properties.—Symptoms.—Period of invasion.—Fatal dose.—Fatal period.—Treatment.—Post-mortem signs.—Diagnosis.—Chemical analysis.—Bitter taste.—Color test.—Fallacies.—Interferences.—The Frog Test.—Recovery from organic mixtures and contents of the stomach.—Detection in the tissues, blood, and urine.—Failure to detect.—Brucia.—Properties.—Tests 396

CHAPTER XXV.

ORDER III.—Cerebro-spinal Neurotics.—Deliriants 436

SECTION I. Poisoning by Belladonna.—Symptoms.—Character of the delirium.—External application.—Atropia.—Fatal dose.—Treatment.—Morbid lesions.—Chemical analysis.—Detection in organic mixtures.—Physiological test 436

PAGE

SECTION II. Poisoning by Stramonium. — Properties. — Symptoms.—Post-mortem appearances.—Analysis.—Daturia . . 444

SECTION III. Poisoning by Hyoscyamus. — Properties. — Symptoms.—Idiosyncrasies.—Analysis.—Hyoscyamia 447

SECTION IV. Poisoning by Solanum. — Solanum dulcamara, or Bittersweet.—Solanum nigrum, or Garden Nightshade.—Solanum tuberosum, or Potato.—Solania.—Tests 449

CHAPTER XXVI.

Depressants 452

SECTION I. Poisoning by Hemlock. — Properties.—Post-mortem appearances.—Conia. — Treatment. — Chemical properties.—Tests. — Detection in organic mixtures. — Other species of Hemlock 452

SECTION II. Poisoning by Tobacco.—Symptoms.—External application.—Morbid lesions.—Nicotina.—Properties.—Case of the Count Bocarmé.—Chemical reactions.—Separation from organic mixtures and from the stomach.—Poisoning by Lobelia.—Lobelina.—Effects on the system 457

SECTION III. Poisoning by Aconite.—Properties.—Symptoms.—Morbid lesions. — Aconitia. — Properties. — Treatment.—Detection in organic matters 465

SECTION IV. Poisoning by Calabar Bean.—Properties.—Effects on the system.—Treatment.—Physostigmia.—Chemical properties 471

CHAPTER XXVII.

Asthenics 474

SECTION I. Poisoning by Hydrocyanic (Prussic) Acid.—Properties. — Symptoms. — Rapidity of invasion. — Fatal dose. — Treatment. — Post-mortem appearances.—Tests.—Process by distillation.—Quantitative analysis.—Cyanide of Potassium. — Oil of Bitter Almonds. — Cherry-Laurel-Water. — Nitro-Benzole, or Essence of Mirbane 474

SECTION II. Poisoning by Digitalis.—Symptoms.—Post-mortem signs.— Digitaline. — Chemical reactions. — Detection in organic mixtures. — Physiological test. — Case of De la Pommerais 490

SECTION III. Poisoning by Cocculus Indicus.—Symptoms.—External application.—Picrotoxia.—Chemical reactions.—Separation from organic mixtures.—Poisoning by Laburnum.—Yew.—Privet.—Guelder Rose.—Holly 496

MANUAL OF TOXICOLOGY.

CHAPTER I.

PRELIMINARY CONSIDERATIONS.

TOXICOLOGY—from τοξικὸν, a *poison*, and λόγος, a *discourse*—signifies the science which treats of Poisons. It embraces the consideration of their nature, properties, history, mode of procuring, effects on the animal system, fatal dose, antidotes, and means of detection by chemical analysis and by other methods.

The subject of Poisoning very properly occupies a prominent place in medical jurisprudence, since in a large majority of the cases of violent death that claim the attention of the legal physician, the fatal result is directly attributable to poison. Before proceeding to treat of the individual poisons in detail, it will be proper first to discuss some preliminary points of a general character, the correct understanding of which is of the utmost importance to the toxicologist, and indeed, without which knowledge, he will be liable to fall into serious errors, when, on the witness-stand, he undertakes to enlighten the court and jury in some capital case. We would, therefore, at the outset, urge upon the student of Toxicology not only the importance of a thorough acquaintance with the chemical methods employed for the detection of the different poisonous agents, but also the necessity of a close observation of all the surrounding circumstances, by which alone he will be enabled to solve the difficult questions that present themselves in nearly every case of suspected poisoning.

Among these questions, the following offer special claims to notice: the inferences to be drawn from the origin and progress of the symptoms that have attended the suspicious case; the character of the post-mortem lesions, when the case has terminated fatally; the proper mode of conducting the autopsy; the value of the signs afforded by experiments upon the lower animals, as tending to strengthen or to invalidate the suspicion of poison derived from other sources; and finally, the necessity of a rigorous observance of the rules laid down for a strict and impartial chemical analysis.

Besides the above considerations, there are others that will not escape the attention of the educated toxicologist. Thus, he will give due attention to the conduct and deportment of the persons in attendance upon the patient; he will not fail to notice any unnatural solicitude about the removal of articles of medicine or food, or of the matters ejected from the stomach and bowels; any undue anxiety to exclude all others from waiting upon the patient; and, in case of death, a disposition to avoid a post-mortem examination, and to hasten the interment of the body with an undue haste. These different subjects will therefore receive a proper consideration, together with a study of the various circumstances which modify the effects of poisons on the human system, such as age, habit, idiosyncrasy, disease, quantity of the poison, condition of the stomach, etc.

A Poison may be defined to be *a substance capable of producing noxious and even fatal effects upon the system, no matter by what avenue it be introduced; and this, as an ordinary result, in a healthy state of the body, and not by a mechanical action.* It will be observed that the above definition of a poison is carefully worded, in order to convey a correct idea of its meaning. Thus, the poisonous effect must be *an ordinary result* of its administration. A substance which affects one person in consequence of some idiosyncrasy or peculiarity of constitution, but does not affect others, is not a poison. The most simple articles of food may act poisonously upon certain individuals, through this idiosyncrasy; we have known a case in which strawberries produced this effect habitually; and the records of medicine are full of such instances. Again, to constitute a substance a poison, its noxious effects must be produced *on the healthy system*. It is well known that many diseased conditions of the body render it extremely susceptible to impressions by external agents, even those otherwise the most harmless. Thus, in acute inflammation of the stomach, almost any substance—even water—may excite vomiting and pain; so also, the copious drinking of cold water by a person heated by violent exercise may occasion sudden death; yet water is not a poison. In a certain diseased condition (granular) of the kidneys, it has been found that calomel, even in small doses, proves very injurious; yet calomel is not ordinarily regarded as strictly a poison. Again, in order to bring a substance within the strict definition of a "poison," it must not act *mechanically*. Certain bodies, such as pins and needles, powdered glass, fragments of iron and other metals, and the stones and seeds of fruits when swallowed, may cause serious and even fatal results by their mechanical action, producing irritation and inflammation of the lining membrane of the alimentary canal; but these are not poisons.

According to our definition, a poison produces its deleterious effects upon the system, *no matter by what avenue it be introduced*. Most usually, poisons are swallowed into the stomach, and are thence absorbed into the circulation. But they act often with far greater vigor when inhaled into the lungs in the form of vapor, when injected into the rectum, when introduced hypodermically, or when applied to an ulcerated or abraded surface. They are also absorbed, though not so rapidly, from the mucous membrane of the vagina, the nose, and the ear, and even from the sound skin. It is to be understood that in all of the above instances, the poison produces its peculiar effects by being absorbed into the circulation; and the rapidity of its effects will always be, *cæteris paribus*, in proportion to the rapidity of its absorption. It is for this reason that when a poison is directly injected into the blood-vessels, its action is so immediate.

The question of *quantity*, or size of the dose of the poisonous substance, does not enter into the account in case of an indictment for poisoning. In medico-legal inquiries, the main object of proof is to connect the results—*i.e.*, the *symptoms*, *lesions*, and *chemical analysis*—directly with the substance employed, and with the *intention* of the person employing it. Thus, the law makes no distinction between a murder committed by the administration of a grain of strychnia and one resulting from taking an ounce of oxalic acid, provided both were given with the same evil design; each is equally fatal, although they differ so widely in their fatal dose. There are some substances, such as sulphate of magnesia (Epsom salt) and chloride of sodium (common salt), which are generally regarded as innocuous, but which have occasioned death in several instances where very large doses have been taken. Dr. Taylor (On Poisons, Am. ed., 1859, p. 19) mentions the case of a young lady who died in the course of a few hours in consequence of swallowing about half a pound of common salt as a remedy for worms. General paralysis set in in about two hours, and all remedies, including the stomach-pump, proved unavailing. The post-mortem appearances indicated excessive irritation of the alimentary canal. Sir R. Christison mentions an instance in which a man died, after swallowing a pound of this salt, within twenty-four hours, with all the symptoms of irritant poisoning; also another case, in which two ounces of the same salt produced very alarming symptoms in a young man. This author also quotes a case of a boy ten years old, who died within one hour after swallowing two ounces of Epsom salt, administered as a remedy for worms; violent symptoms immediately came on, such as great depression, slow and difficult breathing, pulse almost imperceptible, but no vomiting or purging. It is further stated that after death, no morbid appearances were observed in the body. (Christison on Poisons, p. 657.)

From the above remarks it will be evident that the mere *quantity* of the substance required to destroy life cannot be made a ground of distinction between a poisonous and a (so-called) non-poisonous article. In the language of Dr. Taylor (*loc. cit.*), "if a medical witness be asked, What is a poison? he must beware of adopting this common definition, or of confining the term 'poison' to a substance capable of operating as such in a *small* dose given at once."

The term *deadly poison* is popularly applied to such virulent substances as destroy life very speedily and when taken in small doses, such as prussic acid, strychnia, arsenic, nicotina, etc. In the wording of an indictment for poisoning, it is customary to describe every poison as *deadly*, although, in the strict sense of the term, many fatal poisons cannot fairly be thus described. The question, however, may now be considered as settled by judicial decision that the word "*deadly*" is not essential to the validity of an indictment (Law Times, April 12, 1845); its use, therefore, in this connection may be regarded as superfluous.

If the substance administered with criminal intent is capable of destroying the health or the life of an individual, the law makes no account, so far as the responsibility of the prisoner is concerned, of the *manner* by which life was destroyed,—whether by chemical or mechanical means. In order, however, properly to include all the possible methods, the English statute runs thus: "Whoever shall administer, or cause to be taken, any poison or *other destructive thing*, with intent to commit murder, shall be guilty of felony, and being convicted thereof, shall suffer death." Here the phrase "*other destructive thing*" is intended to cover the whole ground, and to preclude the possibility of escape through a faulty indictment. The laws of the United States in relation to the administration of poisons are essentially the same as those of Great Britain.

In relation to the dangerous results which not unfrequently follow the swallowing of irritating bodies (as already mentioned), such as powdered glass, pins and needles, etc., which act only mechanically, it should be remembered that many small substances, such as the seeds of various fruits, are liable to cause death by entering into the *appendix vermiformis* of the cæcum, and there exciting a fatal peritonitis. In such cases the effects may be erroneously ascribed to an irritant poison; and the real cause of death will be revealed only by a post-mortem examination.

Further, certain substances, when swallowed, may produce alarming, and even fatal, effects, by simply over-distending the stomach. A case is mentioned by Dr. Taylor of a young woman who took a quantity of raw rice mixed with milk. She soon became alarmingly ill, suffering with symptoms of over-distension of the stomach. She was much relieved for the time by an emetic; but on the following day the pain and anxiety returned, and she died in twenty-four hours after swallowing the rice. A post-mortem examination revealed an extensive recent peritonitis. It occasionally happens that a person dies from over-distension of the stomach occasioned by eating a hearty meal. In such cases, the actual cause of death is to be ascribed either to apoplexy (arising from the distension), or to shock. Such an accident may happen at any age; and as the symptoms come on suddenly after eating, and are of a very alarming character, the suspicion of poisoning may very naturally be excited, and the real cause of death will be discovered only by a proper examination of the body.

CHAPTER II.

MODE OF ACTION OF POISONS ON THE ANIMAL ECONOMY.

THIS action is of a twofold character: (1) local, and (2) remote.

1. *Local Effects of Poisons.*—By this is understood the direct impression of a poison upon that part of the body with which it comes into immediate contact. The effects thus produced are, for the most part, of an irritant or corrosive character. The strong mineral acids and alkalies, for example, if applied externally, or taken internally, by virtue of their chemical affinities exert a destructive or corrosive action upon the tissue to which they are applied; and they may prove fatal either by the shock occasioned to the nervous system (precisely as in the case of a severe superficial scald or burn), or else, through their destructive agency, by causing a perforation of the stomach or intestines, and thus bringing on fatal

peritonitis. Frequently the local effect of a poison results merely in *inflammation* of the part, which, however, may proceed to suppuration, ulceration, and gangrene. Examples of this are afforded in the action of such irritants as arsenic, tartar emetic, salts of copper and zinc, cantharides, and numerous vegetable substances, all of which produce a local irritant impression when taken internally, or when applied externally; although some of them may at the same time exert a general or constitutional impression, as *e.g.* arsenic and tartar emetic. It will be observed that the action of the local poisons above alluded to closely resembles that of mechanical irritants, except that these latter exert no local *chemical* effects.

The local effects of another class of poisons appear to be especially directed to the sensory nerves, producing a benumbing or paralyzing sensation. Thus, aconite root, when chewed, occasions a pricking, benumbing feeling to the tongue and fauces; chloroform, prussic acid, and veratria, when applied to the skin, produce a sensation of numbness; opium, prussic acid, and ticunas, if applied directly to the muscles, occasion paralysis; and belladonna and Calabar bean produce the same influence on the muscular fibre of the iris when directly applied to the eye,—the former dilating the pupil, and the latter contracting it.

2. *Remote Effects of Poisons.*—By this we understand those results which are produced in parts of the system remote from that to which the poison was originally applied. The same substance, however, often exerts both a local and a remote action, as already observed.

The remote effects of poisons are twofold—common and specific. Their *common* effect is the same as that which would result from any common severe injury inflicted upon the part; their *specific* effect is that which the poison alone could produce. Moreover, the remote specific effect may be either *general*—producing a general specific impression on the whole frame, as *e.g.* the general depression caused by tartar emetic, or *local*, as *e.g.* the local action of tartar emetic upon the lungs and skin, or that of mercury upon the salivary glands.

A proper understanding of the *remote* effects of poisons constitutes the really important problem to be determined: it is that which will indicate the poison itself. Thus, a gradually increasing stupor is a very significant indication of opium-poisoning; tetanic spasms are strongly suggestive of strychnia; salivation, with fetid breath, points to mercury; certain forms of paralysis are symptomatic of lead or mercurial poisoning, etc. These remote effects, or manifestations, in fact, constitute a most important class of *symptoms* of poisoning—a very valuable factor in aiding us to arrive at a positive conclusion in a suspected case, but one, nevertheless, which must be cautiously employed, since there are many diseases whose symptoms very strongly resemble those of poisons (*vide post*).

In order properly to understand the remote action of poisons, the question must first be determined—How do poisons gain access to the different organs of the body after being swallowed into the stomach, injected into the rectum, inhaled into the lungs, or introduced hypodermically into the cellular tissue? This question will be discussed in the succeeding section.

SECTION I.

THE DIFFERENT MODES BY WHICH POISONS GAIN ACCESS TO THE VARIOUS ORGANS OF THE BODY.

There are only three conceivable means by which the influence of poisons can be transferred to distant organs, viz., (1) by propagation of their impression by nervous communication to the great nerve-centres; (2) by contiguity of structure; and (3) by their absorption into the circulation. These three methods of transfer will now be examined.

1. *Nervous communication.—Sympathy.*—In the early history of Toxicology all poisons were believed to act through sympathy. This was also the early doctrine with respect to medicines; in both instances, the impression made on a part of the body by the immediate contact of the substance was supposed to be conveyed to distant parts by means of nervous communication, either directly, or by reflex action through the nervous centres. The discovery of venous absorption

by Magendie, in 1809, entirely revolutionized the ideas of physiologists on this subject. The present doctrine is in favor of the almost exclusive absorption of both medicines and poisons. Of the *fact* of the propagation of impressions by nervous communication, there can be no doubt. Familiar illustrations will readily occur to the practitioner of medicine, as where, in disease, distant organs are affected by sympathy; so that there would seem to be no reason, *a priori*, why the morbid impression produced by a poison or a medicine should not be transferred in a similar manner. Indeed, the extreme rapidity with which certain poisonous agents, when locally applied to the body, affect the centres of life, rather countenances this mode of action; whilst the same velocity appears inconsistent with the idea of its action through the route of the circulation; thus, a drop of pure prussic acid applied to the tongue of a cat, killed it almost instantly—within three or four seconds. But the subsequent experiments of Blake and Hering, by demonstrating the extreme rapidity of the circulation, have served to reconcile the instances of the most speedy action of certain poisons with the theory of absorption. It is, however, undeniable in some cases, *e.g.* those of rapid death from the action of the corrosives, and where the fatal result is occasioned by what is denominated shock, that the impression is conveyed from the point of injury, through nervous communication, to the great nerve-centres.

2. *Contiguity of structure.*—Very little need be said on this subject. With the old writers this was a favorite method of accounting for the propagation of morbid impressions. It was subsequently extended so as to embrace the action of remedial and toxic agents; but at the present day very few, if any, authorities sustain it. The Absorption of poisons will be discussed in the following section.

SECTION II.

ABSORPTION OF POISONS.—CIRCUMSTANCES INFLUENCING ABSORPTION.—SUBSEQUENT DISPOSITION OF THE POISON.—ELIMINATION OF POISONS.—CAUSE OF DEATH.

3. *Absorption of Poisons.*—This is by far the most important of all the methods by which poisons act upon the body. The fact that poisons are absorbed into the circulation after being swallowed, or otherwise introduced into the body, is fully established by numerous experiments, both upon man and the lower animals. The *proofs* of absorption are afforded by their detection in the blood, in the secretions, and in the different organs of the body; and the list of poisons thus detected includes every substance which can be recognized by its color or odor, or by chemical analysis. Tiedemann and Gmelin discovered acetate of lead in the blood of the splenic and other veins of dogs; and cyanide of mercury and chloride of barium in the blood of the vena portæ and of the splenic vein of the horse. Wöhler detected, in the urine of dogs and horses, iodine, sulphuret of potassium, nitrate of potassa, sulphocyanide of potassium, the salts of nickel, and oxalic, tartaric, citric, malic, gallic, succinic, and benzoic acids. Orfila found arsenious and arsenic acids, the arsenites, the soluble arsenates, tartar emetic, iodine, potassa, and its salts, the salts of baryta, the mineral acids, ammonia, muriate of ammonia, and the soluble salts of lead, copper, mercury, gold, and silver. He equally detected the poison in the blood, whether it was swallowed into the stomach, or applied externally (Toxicologie, 1852, i. p. 18).

Since the time of Orfila, the advance in chemical analysis has further extended the list of poisonous substances that have been detected in the blood, the secretions, and the tissues and organs of the body. Even the organic poisons (alkaloids) prove no exception to the rule, as many of these have been discovered in the blood and urine, and a few even in the solids of the body. The recognized duty of the toxicologist of the present day is not merely to discover the poison in the contents of the stomach, but also to find it, *in the absorbed state*, in the different viscera of the body, or else offer a satisfactory reason for not finding it there. (See *post*, ELIMINATION OF POISONS.)

The detection of a suspected poison before death in the *urine*, and after death, in this and in other secretions, and particularly its discovery in the organs of the body, affords the most unequivocal proof of the administration of the poison before death.

The absorption of poisons is influenced by a variety of circumstances: (1) *Solution.*—In order that absorption should take place, it is necessary that the substance should be in a state of solution: insoluble bodies are not absorbed. Many substances, however, when swallowed in an insoluble condition, may afterwards become soluble, by virtue of certain chemical agencies set up in the stomach, and then be absorbed.

To the general proposition that insoluble bodies are not absorbed, there seems to be an exception in the case of finely powdered wood-charcoal. Prof. Oesterlen, of Dorpat, and Mensonides, of Utrecht, assert that they discovered charcoal in the veins and in various organs of animals fed upon this substance several days before. MM. Mialhe, Lebert, and Bernard were, however, not able to verify their results; subsequently, MM. Orfila, Robin, and Bérard repeated the experiments of feeding dogs on charcoal for several days, and then killing them by hanging. By means of a microscope of 450 power, they were able to discover molecules of the charcoal in the blood of the liver and of the lungs, in that of the left auricle, and in a mesenteric gland; but not in the blood of the vena portæ, nor in the chyle. In a subsequent experiment with *lampblack*, performed in the same manner, they were unable to discover the slightest trace of the carbon in any part of the animal's body. Orfila's conclusion was that when particles of charcoal passed into the blood from the stomach, it was owing to the fact of their being "exceedingly sharp-pointed and angular (as is the case in the charcoal of wood, but not in lampblack), by which means they forced a passage through the delicate capillaries." (Toxicologie, 1852, i. p. 25.)

(2) *Nature of the surface to which it is applied.*—The rapidity of the poisonous impression is in direct ratio with the absorbing power of the part to which it is applied; and this is chiefly dependent on its supply of blood, or its vascularity: this is easily understood. Hence, when directly introduced into the circulation, by injection into a vein, the impression is the speediest of all. Sir R. Christison found that when the muriate of conia was injected into the femoral vein of a dog, he was unable, with his watch in his hand, to notice any appreciable interval between the moment at which it was injected and that at which the animal died; certainly the interval did not exceed three or four seconds. (On Poisons, p. 8.) Other poisons injected in the same manner will act with equal celerity. Next in order comes inhalation, in the form of vapor, into the lungs. From the extreme vascularity of the pulmonary air-cells, a vapor-substance introduced into these immediately finds its way into the circulation. Next, the cellular or areolar tissue affords a very speedy mode of introducing a poison by hypodermic injection. The serous membranes come next in order; then the stomach and bowels; and last of all, the sound skin.

In certain instances, a poison introduced into the rectum acts more promptly than when taken into the stomach, although these cases are exceptions. This is asserted to be true of arsenic, corrosive sublimate, strychnia, and the preparations of opium.

In regard to some of the *animal* poisons, the mucous membrane of the stomach appears to exercise a remarkable modifying influence over them, inasmuch as they may be swallowed with impunity even in large doses, while the smallest fragment of them introduced beneath the skin of the same individual produces rapidly fatal results. Notable illustrations of this fact are furnished in the case of the virus of poisonous snakes and of the mad dog, and that of glanders. Other animal viruses, such as the matter of variola and syphilis, are likewise innocuous when swallowed, but produce, as is well known, their specific effects very speedily when inoculated beneath the skin. This is also true of the *woorara* poison.

The absorption by the stomach is modified by the full or empty condition of that organ,—being most rapid when the stomach is empty. It is doubtless for this reason, that persons frequently escape death after swallowing large doses of a poison on a full stomach.

The *sound skin* may, in some cases, become the avenue for the introduction of poisons by absorption, as seen in the cases of arsenic, corrosive sublimate, sugar of lead, opium, and many other substances which have occasioned serious and even fatal consequences when thus applied. Moreover, after death they have been detected in the tissues of the body—showing that they had been absorbed. When the cuticle is removed and the poison is applied directly to the true skin (*endermically*), absorption is much more rapid. Hence an ulcer, or a wound, is a ready medium for the absorption of poisons.

(3) *Fullness of the blood-vessels.*—The rapidity of absorption is always inversely to the quantity of the circulating fluid. The fullness of the blood-vessels opposes a mechanical obstacle to the entrance of any other fluid. Hence depletion of the vessels by bleeding or purging will favor absorption. For this reason, in a case of poisoning, it is generally considered injudicious to bleed the patient,—the loss of blood increasing the further absorption of the poison from the stomach into the general circulation.

But admitting the fact of absorption, the question arises,—Is the fatal effect of the poison to be ascribed to this? This question must be answered in the affirmative, if it can be shown, on the one hand, that poisons continue to act so long as the blood passes freely from the point of insertion to the tissues or organs affected, and that, on the other, their action is stopped or postponed when the circulation is arrested.

The oft-mentioned experiment of Magendie establishes the first of these propositions. He divided the leg of a frog from the body, and established a connection between the separated parts by means of quills inserted into the large vessels. Nux vomica was then applied to the foot, when absorption took place, and death resulted with the characteristic symptoms of that poison.

The second proposition is proved both by the foregoing experiment, and by one of Mr. Blake's: Prussic acid was introduced into the stomach of a dog, through an opening in its walls. No poisonous effect was produced so long as the vessels passing from the stomach to the liver were secured by a ligature; but it began to act within one minute of its removal (Ed. Med. and Surg. Jour., Jan., 1840).

The *rapidity* of the absorption is remarkable. As the result of numerous experiments upon animals, it has been proved that a poison injected into the cellular tissue will be diffused throughout the whole circulation in a few seconds, and a solution of sulphuretted hydrogen in water injected into the rectum of a dog, passed through the circulation and was eliminated by the lungs in sixty-five seconds (Bernard, Leçons, p. 59). The rapidity with which absorption goes on will, as before remarked, satisfactorily account for the mode of action of even the most promptly fatal poisons, without the necessity of resorting to any other theory to explain it.

Subsequent disposition of the Poison.—After the poison has entered the circulation, it may either be rapidly eliminated by the different emunctories, especially by the kidney, or it may be temporarily deposited in the organs and tissues of the body, and usually in the following order as to quantity: the liver, spleen, kidneys, heart, lungs, brain, and pancreas. Experiment has shown that only a minute quantity of the poison is circulating in the blood *at any one time;* the effort of the system evidently being to get rid of it as rapidly as possible. Moreover, there is good reason to believe that the poison is active *only while circulating in the capillary blood-vessels:* while still in the stomach, or after separation from the blood by the emunctories, or when deposited in the solid tissues, it is believed to be entirely harmless. It is a very common mistake to suppose that in a fatal case, death is caused by the very poison discovered *in the stomach;* whereas this has no actual connection with the fatal result,—the death being, in point of fact, attributable to the *absorbed* portion only (except in the case of the corrosives). The quantity remaining in the stomach after death is merely the complement of the fatal portion,—the surplus of what was necessary to kill. The same is true of that portion of the poison which has been deposited in the liver and other organs of the body: it has been removed, for the time being, out of the sphere of noxious power. But although harmless so long as retained in this situation, it should be remembered that the poison is liable to be reabsorbed into the circulation, when it will again become active. We have no proof that *all* poisons are deposited in the organs: while the fact is true generally of *metallic* poisons, and of some of the organic poisons (alkaloids), it has not yet been fully demonstrated in the case of all. The gaseous poisons, as sulphuretted hydrogen, appear to be immediately eliminated by the lungs without being deposited in the organs. The experiment of Bernard, quoted above (p. 26), proves this assertion. Another experiment of his still further establishes it: he injected into the jugular vein of a dog one-quarter of a cubic inch of water saturated with sulphuretted hydrogen, the vein being secured above to prevent the escape of blood, and the liquid being gently propelled towards the heart. A piece of paper wetted with acetate of lead solution was held to the dog's mouth; it was blackened in from three to five seconds, showing that the gas had been eliminated from the lungs. This elimination was completed in a few seconds (Leçons, p. 59).

The period when an absorbed poison begins to be removed from the circulation, either by elimination, or by deposition in the organs, varies for different substances, and probably also for different states of the system, for different ages, and even for the different sexes. As regards medicinal substances, it is well known that they appear in the urine in a very short time after being swallowed,—*e.g.* iodide of potassium in ten minutes, and ferrocyanide of potassium in from one to thirty-nine minutes (Erichsen, Med. Gaz., xxxvi. p. 363). It has also been found that the *rate* of elimination, like that of absorption, is by no means uniform. In relation to the mineral poisons, experiments on animals show that *arsenic* may be diffused throughout the body in *one hour and a half* after being introduced. In an experiment by Orfila, three grains of solid arsenic were applied to the cellular tissue of the back of a dog; the animal vomited in half an hour, and died in *four* hours. Arsenic was found, on examination, in the liver, spleen, kidneys, lungs, heart, brains, alimentary canal, and muscles (Toxicologie, 1852, i. pp. 381, 383). This same poison has been found in the urine of a horse within an hour after administration.

Dr. Taylor has found arsenic in the human liver in so short a period as *four hours*, and in another case, in *six* hours, after being swallowed, — the cases having proved fatal in these periods. He thinks that the *liver* acquires its maximum saturation in about *fifteen hours*. He gives a table of an estimate of the average amount of arsenic that will be found in the human liver at different periods of the examination, as follows: In 5½ to 7 hours after taking, the quantity found is 0.8 grain; in 8¾ hours, the quantity is 1.2 grs.; in 15 hours, the quantity is 2.0 grs.; in 17 to 20 hours, the quantity is 1.3 grs.; in 14 days, the quantity is 0.17 gr. (On Poisons, 1859, p. 116). Orfila's opinion was that arsenic is entirely eliminated from the human system in from twelve to fifteen days. Dr. Maclagan treated a case which recovered from a large dose of arsenic. The poison was detected in the urine from the second to the twenty-fifth day, when it entirely disappeared (Ed. Month. Jour., 1852, p. 131). The case of Dr. Alexander, who lived for sixteen days after taking, unknowingly, a large dose of arsenic, confirms Orfila's opinion in relation to the period required for its total elimination from the human body. In this case, a careful examination after death failed to detect the slightest trace of the poison,— showing that it had in sixteen days entirely disappeared by elimination.

The experiments of M. L. Orfila have given us the fullest information upon this subject. From these it would appear that for arsenic and corrosive sublimate, thirty days are required for complete elimination; for tartar emetic, four months; for nitrate of silver, five months; for acetate of lead and sulphate of copper, over eight months (Tardieu sur l'Empoisonnement, p. 19).

The question of the elimination of poisons may assume a serious importance in a medico-legal case, as when there is a failure to discover the poison after death in a person who has died within a few days after the alleged administration of arsenic, and after exhibiting symptoms consistent with this poison. Here, the defense would strongly insist that if the poison had really been taken, it could not be entirely eliminated from all the organs of the body in two or three days, and therefore the failure to detect it *in the absorbed state* was a proof of its non-administration. Dr. Taylor alludes to a case which he examined, where arsenic had caused death in *twenty-six hours;* the poison had nearly disappeared from those parts of the body where it is usually found. There had been much vomiting and purging previously. Hence it would not be safe, in trials, to push the above rule in relation to the period of elimination, too far.

In the case of a person who had been taking arsenic in small doses, medicinally, for some weeks or months before death, and who had died suddenly under suspicious circumstances, the chemical examination might reveal the presence of the poison in the liver. This fact would probably be regarded as positive proof of criminal administration, especially if it could be shown that the deceased had not taken the medicine for upwards of fifteen days before death; and consequently, that the poison found in the liver could not be ascribed to the arsenic which he had taken medicinally. We think it would be very unsafe to admit this plea, under the existing circumstances, since it is quite possible that the elimination in this case might not have been so rapid as is usual; and again, because we have positive proof that arsenic has been detected in the urine of a person as late as the *twenty-fourth day* after it ceased to be administered. If, however, in the above case, *free* arsenic had been discovered in the contents of the stomach, especially in any notable amount, then there could be little doubt of the fact of poisoning. Yet even in such a case it would not amount to *positive* proof, as will be seen on further consideration. (See *post.*)

The case may be presented under a different phase—as where the criminality of the accused is made to depend upon fixing the time required for depositing a certain amount of arsenic found in the liver of the deceased.

Thus, in a certain case reported by Dr. Taylor (*op. cit.*, p. 53), a woman was accused of killing her husband with arsenic. The duration of his illness was about seventeen hours; and the question was whether he had taken the poison by mistake in the morning, or whether his wife had given it to him later in the day. Dr. Letheby stated, in evidence, that he had discovered eight and a half grains of arsenic in the stomach, and two grains (estimated) in the liver; and he gave as his opinion that the poison could not have been taken more than *two or three hours before death*,—based upon the quantity thus discovered. The prisoner was convicted and sentenced to death; but a timely interference by distinguished experts satisfied the authorities that an error had been committed, and that the inference of Dr. Letheby as to the *time* of the administration was unwarrantable. It will be recollected that the average time of maximum saturation of the liver (about two grains) is stated to be *fifteen hours.* (See p. 28.)

In a French case reported in "Ann. d'Hyg. et de Méd. Lég.," 1846, p. 149, the conclusion arrived at was directly the opposite of the one just mentioned, viz., that arsenic had been taken only a few hours before death, because the liver contained *no trace of the poison!* This was much more in accordance with experience.

The question of *elimination of poisons* will be again referred to when treating of the individual poisons.

Cause of Death.—The next question to determine is—After the poison has reached the different parts of the body by means of the circulation, *how* does it produce its peculiar effects? How does it destroy life? Much of our knowledge upon this point is still speculative; nevertheless, experiments on the lower animals, conducted with proper caution, have led to some definite results. In all cases of acute poisoning, where the symptoms run their course rapidly, life is destroyed through an impression either upon the heart, the lungs, the brain, or the spinal marrow; but *why* the poison acts upon one of these great centres in preference to another is a question that brings us to one of the "ultimate facts" of science, beyond which we cannot advance. To say that the poison (and the same is true of medicines) has an affinity for this or that particular organ or tissue, is evidently no explanation of the action. All we can know is the simple *fact*, and the conditions under which the action takes place. But it is an important point to be able to determine these *conditions* and the circumstances attending them. To some of these we shall now give attention.

We start with the admitted fact that poisons enter the circulation, and are thus carried to the different organs of the body, which, as we have seen, are differently affected by them. Some, carried to the heart by the coronary arteries, paralyze that organ; others act directly on the lungs, causing suffocation by arresting the capillary circulation; a third class attack the brain, producing fatal coma; a fourth impress the spinal marrow, exciting fatal tetanic spasms in the respiratory muscles; and a fifth appear to affect the entire capillary circulation. (Guy's Forensic Medicine, 1868, p. 337.) The knowledge of the above facts is derived chiefly from the experiments of Mr. Blake and of Claude Bernard (Med. Times and Gazette, 1860). Moreover, in some cases the same poison will affect different organs according to the dose, and probably also according to the constitution of the poisoned subject. Thus, arsenic, while ordinarily spending its power upon the stomach and bowels, will sometimes affect the heart—as indicated by syncope; sometimes the brain—as shown by coma; and sometimes the spinal marrow—as seen by the tetanic convulsions, numbness, and paralysis that are occasionally manifested. The same is true to a certain extent of oxalic acid, antimony, and some other substances.

One mode in which death occurs by poisoning is undoubtedly by *shock* on the general nervous system. In this way the active corrosives most probably prove fatal—their powerful local action causing a general depression of the system, similar to that occasioned by any severe injury. The *nature* of this fatal shock cannot be determined by any means at present at our command. As in sudden death from concussion of the brain, there may be no external injury, nor any perceptible internal lesion, to reveal to us the actual cause of dissolution; although it is highly probable that it is due to some molecular disturbance of the great nerve-centre.

The fact that most poisons enter the circulation before reaching distant organs, naturally suggests the hypothesis that they first produce some alteration in the character of the blood—chemical or physical,—the poison itself also undergoing change at the same time. Thus, certain substances are supposed to remove oxygen from the blood, in their transit through the circulation, such as phosphorus and arsenic when associated with hydrogen, oxalic acid, alcohol, chloroform, prussic acid, benzoic acid, hydrate of chloral, etc. Chloroform, for example, when swallowed, passes into the circulation, but is obtained from the blood by distillation, as *formic acid*,—its three atoms of chlorine having been replaced by three atoms of oxygen. There is no question that many medicinal substances do undergo a chemical change in passing through the route of the circulation; but whether this change is effected at the expense of the blood is not so clearly proved. The salts of the vegetable acids (acetates, tartrates, citrates, etc.) are eliminated in the urine as *carbonates*, affording undoubted evidence of a chemical transformation, and that probably while in contact with the blood. That decided chemical changes may take place in the blood, and prove fatal by the production of poisons from inert substances, is proved by the following experiment of Bernard. The emulsine of sweet almonds and the amygdaline of bitter almonds, both inert substances, when mixed together in contact with water, react upon each other so as to produce prussic acid. Bernard injected fifteen grains of amygdaline dissolved in water into the jugular vein of a rabbit; no injurious effects resulted. The experiment was then tried with a solution of emulsine, with negative results. But when the one solution was injected into the vein soon after the other, the animal died from poisoning with prussic acid,—this substance having been formed in the blood. In this way it is supposed that miasmata and other noxious causes of disease may operate, by getting into the circulation, after being inhaled into the lungs; and when in the blood, generating the poison which proves so detrimental to life. Bernard ascribes these changes to a species of fermentation (Leçons, p. 96).

Liebig's theory (at least with respect to the action of the poisonous alkaloids) was that they entered into chemical combination with the nerve-substance,—morphia with brain-substance, for instance,—and thus the quality of the nervous matter being altered, it became unfitted to support life. Another theory is that poisons act by destroying the vitality of the blood. But, as is justly remarked by Prof. Taylor, this destruction of the vital properties of the blood does not explain the specific differences of poisons, seeing they do not all act alike.

As regards any actual alteration *in the blood itself*, — either chemical or physical, — nothing has been yet satisfactorily demonstrated, except occasional changes in its color, consistence, and coagulability. Microscopic observation has failed to show any alteration in the appearance of the blood-corpuscles that can be regarded as conclusive.

CHAPTER III.

CIRCUMSTANCES WHICH MODIFY THE ACTION OF POISONS.

THESE may be considered under the three heads of—(1) such as relate to the poison itself; (2) such as are connected with the part to which they are applied; and (3) those which depend upon the condition of the body.

1. Among the modifying causes *connected with the poison itself*, the *quantity* and *form* alone require a brief notice. As a general rule, the larger the *amount* of a poison, the more rapid and powerful are its effects. But there are certain exceptions to this rule. When a very large dose of an *irritant* poison is swallowed, it may be so promptly and completely

rejected by vomiting, as to prevent its fatal action, whereas a smaller dose would have been retained. Again, it would appear from experiments, that certain substances, when swallowed in large doses, seem to produce a local impression on the mucous membrane of the stomach and bowels that interferes materially with the power of absorption, and consequently the impression upon the system must be modified. The effects of some poisons are materially modified by the size of their dose: thus, a very large dose of oxalic acid will kill almost instantly by shock; in a smaller dose, it may still prove fatal by its action on the heart; and in yet smaller doses, it affects chiefly the spinal cord and the brain. In most, if not all, of the mineral poisons, small and repeated doses will develop symptoms very different from those produced by a single large dose.

In relation to the *form* in which a poison is administered, we have already (p. 23) considered the influence of solubility in promoting absorption, and thereby increasing the activity of the poison. The only other point under this head demanding notice is the effect of *combination* or *mixture*. As is well known, two powerful poisons may chemically neutralize each other more or less completely, as *e.g.* the mineral acids and alkalies. Powerful acid poisons in combining with bases, or powerful basic poisons in uniting with acids, produce compounds which, if soluble, retain the characters of the more active ingredient: thus, the soluble salts of oxalic acid partake of the properties of that acid; and the different soluble salts of morphia exhibit the peculiar effects of this alkaloid. In some instances, the chemical combination of two active poisons gives rise to the mixed effects of the two, as in the union of hydrocyanic acid and mercury. The great object in the use of *antidotes*, in the treatment of poisons, is to convert the active noxious substance, by means of a chemical combination, into an insoluble, inert one.

The effect of *mixture* upon poisons is sometimes to increase, and at other times to diminish, their power. Thus, acids increase the activity of opium, and of the alkaloids generally; oily, mucilaginous, and albuminous substances retard the activity of poisons, chiefly by their mechanical influence, protecting the coats of the stomach and enveloping the poison, if in the solid state. Hence the advantage of the free use of such substances in the treatment of irritant poisoning. The subject of *compound poisons*, or the *antagonism of poisons*, will be discussed hereafter.

2. *The modifying influence caused by the part to which the poison is applied.*—As this is dependent simply upon the relative absorbing power of different parts of the body, the reader is referred to what has already been said upon that subject (see *ante*, p. 24).

3. *The influence exerted by the condition of the body itself.*—The conditions of the body that influence the activity of a poison are Habit, Idiosyncrasy, Disease, and Tolerance.

(1) *Influence of Habit.*—It is well known that the effect of habit is to diminish the power of certain poisons. Daily experience demonstrates this in the case of opium, tobacco, and alcohol. It is true of narcotics especially, that the system soon becomes accustomed to their effects, and that it is necessary gradually to increase their dose, in order to keep up the desired impression. The confirmed opium-eater, for example, will take with impunity a quantity of the drug ten or twenty times greater than he could have ventured upon at the beginning without a fatal result. It should not be forgotten, however, that these poisons to which the system seems so easily to adapt itself, produce permanently injurious impressions. This is notoriously true of both alcohol and opium; also of tobacco, though in a less degree. It would appear that the influence of habit is not equally exerted upon mineral poisons. We rarely hear of persons becoming habituated to these enormous doses of corrosive sublimate, arsenic, or tartar emetic,—we mean in the ordinary state of the health.

An apparent exception to this remark is afforded in the case of the arsenic-eaters of Styria. There seems to be well-grounded authority for believing that the Styrian peasants have, from early practice, acquired the habit of swallowing as much as from three to five grains of arsenious acid (white arsenic) at a single dose, and repeating this practice twice a week with perfect apparent impunity (*vide* Dr. Roscoe's paper, read to the Manchester Philosophical Society, Oct. 30, 1860;

also Dr. Maclagan's observations in Chem. News, Lond., July, 1865). It is said that this practice of arsenic-eating is resorted to by the men for the purpose of increasing their physical endurance, enabling them the better to make fatiguing marches up the mountains, and by the women for the purpose of improving the complexion. It is proper to observe that medical experience does not confirm these results in ordinary practice.

The subject may occasionally present itself under a medico-legal aspect, as where the attempt is made by the defense in a trial for arsenic poisoning, to account for its presence in the stomach of the deceased on the ground of his having been an arsenic-eater. It need hardly be observed that such a defense must be worthless, if the symptoms of arsenical poisoning were present before death.

(2) *Idiosyncrasy.*—By this is meant the individual peculiarities of persons. They are almost as varied as the number is great. The effect of idiosyncrasy is usually to render the individual more susceptible to certain impressions than ordinary: thus, to some persons the smallest dose of mercury or of opium will act as a violent poison. In the case of others, articles of food that are generally perfectly harmless, will produce the symptoms of violent poisoning. The instances in which idiosyncrasy produces a diminished susceptibility to the action of poisons are rare. An illustration is afforded in the well-known case mentioned by Sir R. Christison (On Poisons, p. 32), where a gentleman unaccustomed to the use of opium took nearly an ounce of laudanum at a single dose, without any effect.

The subject of idiosyncrasy may become of importance in a medico-legal point of view, as where symptoms of poisoning follow a meal consisting of a particular kind of food. If other circumstances should happen to favor the suspicion, the most serious error might be committed in attributing to poison what was really due to another cause. (See *post.*)

(3) *Disease.*—The effects of disease in influencing the activity of poisons are displayed in two opposite conditions: it sometimes increases, and at other times diminishes, the susceptibility of the system to the impression of a poison. Instances

of the former are witnessed in apoplexy and inflammation of the brain, causing a greater susceptibility to the action of opium and other narcotics; also in certain diseased conditions of the kidney, producing an increased susceptibility to the impression of mercury. As examples of the latter, we may cite the greatly diminished susceptibility to the action of opium in tetanus, mania, and delirium tremens. In inflammation of the stomach and bowels, there is an increased susceptibility to the action of irritant poisons. In paralysis, increased doses of strychnia are borne without bad effect.

A knowledge of the above facts is important in reference to a charge of malapraxis, where an ordinary dose of a medicine, *e.g.* calomel, has produced a poisonous effect on the system, through some diseased state of its organs.

(4) *Tolerance.*—By this is understood the ability of the system to bear very large doses of certain poisonous substances, in consequence of a morbid condition of the economy, and altogether independent of habit. A good illustration is afforded in the case of tartar emetic. In acute pneumonia and bronchitis, and in acute rheumatism, immense doses of this substance have been given, not only without producing its usual poisonous effects, but with a positive mitigation of the disease. This practice originated with Tommasini, Rasori, and Laennec, and was formerly very much in vogue under the name of the Italian, or contra-stimulant system. The facts connected with the tolerance of certain medicines in poisonous doses are of some medico-legal importance, where, for example, in a case of tartar-emetic poisoning, it might be alleged that this substance is not poisonous *because* it has so frequently been used in enormous doses! But the important fact that such quantities are tolerated only in certain morbid states of the system, is studiously kept back.

The influence of *sleep* upon the action of poisons may be briefly noticed here. In this state all the functions are carried on with less activity, and the system is less alive to the impression of poisons. Hence the action of a poison taken at night, just before going to sleep, is very apt to be retarded for some hours. Sir R. Christison alludes to this in the case of arsenic; and there is no reason why it should not hold good with irritant poisons generally. The artificial sleep produced by opium and other narcotics may exert the same retarding influence: thus, according to Prof. Guy, opium, when given with arsenic, not only masks the symptoms proper to that poison, but appears also to retard its operation.

CHAPTER IV.

POST-MORTEM IMBIBITION OF POISONS.

As being closely connected with the subject of the absorption of poisons, it will be appropriate here to consider the question of *imbibition of poisons after death.* Is it possible for a dead body to imbibe a poisonous substance from the soil in which it has been interred? and is it possible that a poison introduced into the stomach or the rectum, or by the hypodermic method, after death, should pass through the tissues by imbibition into other viscera of the body, so as to give rise to the suspicion of poisoning, when in reality the death had resulted from a different cause? These are extremely important questions in a medico-legal point of view, and they deserve to be carefully studied.

In relation to the first proposition, whether a dead body buried in a grave can absorb any poisonous matters from the soil,—the question is narrowed down to the case of arsenic, since this is the only poison about which there is ever any dispute. It is undoubtedly true that arsenic is frequently found in the soils of certain cemeteries; but it never exists there *in a soluble form*, but always in combination with either iron or lime, in an insoluble state. According to the highest authorities, it cannot be extracted from such soils by even boiling water alone, but it requires the action of hydrochloric acid to effect its solution,—consequently it is impossible that it should be capable of transudation from the soil into a dead body.

The only case that could possibly give rise to a suspicion of this sort is where the body has been buried in the earth for so long a period as to have allowed the coffin to become completely disintegrated, and the putrefied mass to lie in immediate contact with the arsenical soil, and the mixed matters are subjected to chemical analysis. But the cases in which this plea has usually been urged have been those of comparatively recent death, where there had been no such contact with the soil; cases, moreover, in which the poison has been detected in the different organs of the body—in their interior as well as on the surface,—whereas, if by any chance the naturally insoluble arsenic could have been made soluble, and entered the body by imbibition, more of it would have been discovered *on the outside* than in the interior, which is quite contrary to the facts of the analysis. Hence we must conclude that the idea about the imbibition of *cemetery arsenic* is an unfounded one; and wherever this poison is discovered after death disseminated throughout the different organs of the body, and particularly if it can be ascertained that the symptoms before death were those of arsenical poisoning, we think there can be no doubt that it had been administered during life. An experiment of Orfila, quoted by Dr. Taylor (On Poisons, p. 378), would seem to settle this matter. He procured a large quantity of earth from a cemetery known to contain arsenic, and buried in it a full-grown fœtus, the liver of an adult, and various portions of dead human bodies. Three months afterwards, these various parts were exhumed, and found to be in a state of complete putrefaction. They were carefully examined for arsenic by the usual processes, but not a trace of the poison could be detected. Hence it appears evident that under the most favorable circumstances, the dead human body does not acquire an impregnation of arsenic from contact with arsenical earth.

The other proposition under this head is, whether it is possible for a poison existing in the stomach at the time of death, or introduced into it after death, to be diffused throughout the body by imbibition, so as to be discovered *in the organs* by chemical analysis. In reply to this we would say that if a poison is discovered very shortly after death in the different organs of a body, it is fair to presume that it was deposited there *during life*, by absorption. Under such circumstances, the viscera will contain as much of the poison *in their interior as on their surface*,—which would not be the case if they had derived it by post-mortem imbibition. But if the poison is found in the organs of a body after many years' interment, then the objection may be very plausibly urged that its presence in the different viscera may be ascribed to imbibition from the stomach and bowels.

Orfila's experiments upon dead human and animal bodies with solutions of arsenic clearly demonstrate that such imbibition does actually take place, but that it is only partial,—affecting mostly only those viscera that were in immediate contiguity with the stomach (see Toxicologie, 1852, i. p. 63). These facts are of practical importance in relation to the proper method of preserving the viscera, in a case of suspected poisoning: the liver and other organs should never be put into the same jar with the stomach and intestines, since the former, although entirely free from the poison, might acquire it by imbibition, and give rise to very erroneous conclusions as regards the absorption and deposition of the poison during life.

There is one aspect of this question which, though an improbable one, is not impossible,—viz., the designed introduction, after death, of a poison in solution, either into the stomach or the rectum, or hypodermically into the cellular tissue, with the criminal purpose of exciting a suspicion of poisoning against an innocent person. Some of the highest authorities admit the *possibility* of this occurrence. Sir R. Christison (On Poisons, p. 61) says: "Although I have not been able to find any authentic instance of so horrible an act of ingenuity having been perpetrated, it must nevertheless be allowed to be quite possible." Orfila evidently contemplated the possibility of such a crime, although he admits never to have heard of its actual occurrence (Toxicologie, 1852, i. p. 61).

Under such circumstances, we must admit the fact of imbibition or osmosis, on well-known physical principles. In a short time the different viscera would become more or less impregnated with the poison, and an analysis might detect it, even in the liver, spleen, kidneys, etc.; and such a discovery would usually be regarded as satisfactory proof that the poison had been administered *during life.* A remarkable case of this character occurred in the State of Ohio, in 1871; it is known as the *Buffenbarger Case.* The deceased was an aged man, who had been treated in his last illness for phthisis pulmonalis; his physician testifying to his having died of this disease, and to his presenting none of the symptoms of arsenical poisoning before death. The body had been interred four years, during all which interval no suspicion of foul play appears to have been entertained. In the mean time the widow married again. After this, for reasons not known to the author, the question of poisoning was raised, the widow was charged with the crime, and the body was exhumed for judicial examination. The autopsy revealed a remarkable state of preservation of the body: "The walls of the abdomen and skin and the subcutaneous tissue were all quite firm and solid. The stomach was not so well preserved (although the organ was entire, and the tissue parchment-like); the liver was much broken down." Arsenic was discovered both in the stomach and liver by Prof. Wormley (the quantity not stated).

The main question was to account for the presence of the poison in the viscera. The defense contended that it had been introduced after death into the body, and distributed to the different organs by *cadaveric imbibition.* The chief points urged by the prosecution were the actual discovery of the poison, and the remarkable state of preservation of the body after an interval of four years.

In relation to the latter fact, while the antiseptic powers of arsenic cannot be questioned, it is equally well established that it does not always prevent even rapid decomposition of a body; on the other hand, there are well-attested instances of a remarkable resistance to putrefaction in bodies, *without* the preserving influence of arsenic. What is the real preservative influence in cases of this character is not fully known. The conversion of the tissues into *adipocere* will sometimes account for it; but this peculiar change does not seem to have occurred in the present instance. It is a significant fact that there is no mention of the presence of any *yellow sulphide* of arsenic in the alimentary canal or elsewhere, which would almost certainly be the case if the poison had been taken during life. Every toxicologist is aware of this transformation occurring after burial, through the agency of the sulphuretted hydrogen of decomposition; and the longer the interval after death, the more likely will it be to take place. Again, the broken-down or pulpy condition of the liver is rather adverse to the theory of ante-mortem poisoning. Arsenic seems, as it were, to have a special affinity for this organ; more of the *absorbed* poison is found in the liver than in any other organ of the body. In our own experience, this organ is always firm and well preserved by the *absorbed* arsenic, even years after death.

We are not in possession of all the circumstances connected with this singular case; although we have reason to believe that the opinion of those most conversant with the particulars was that the poison had been designedly introduced into the body after death, and not very long before the trial, with the view of creating the impression that the man had been poisoned by his wife. A very significant fact is the circumstance that the case had only a preliminary hearing, after which it was abandoned by the prosecution. As this is the first case of the kind recorded, we regard it as of considerable importance as a leading one in this particular line.

Orfila lays down the following rules (among others), as aiding us in the diagnosis of such cases:—1. "When irritating and corrosive substances *in the solid state* are introduced into the body after death, there always remains a very considerable quantity close to the spot to which they were applied, and none is found in the alimentary canal at any distance from this point. This is especially the case if no great interval of time has elapsed, and if the substance has not been dissolved in the contained liquids. On the contrary, in cases where the poison has been administered during life, only a little is found after death, because the most of it has been expelled by vomiting and purging." In relation to the application of this rule, we would observe that, while it is true in general, it does not meet the exceptional cases (quite numerous) where very large quantities of an irritant poison (arsenic) have been found in the stomach, where the death is known to have resulted from poisoning. It is well known that in some anomalous cases of fatal arsenical poisoning, there has been neither vomiting nor purging; and no evidence of irritation or inflammation of the alimentary canal discovered after death. Such cases, according to this rule, might be mistaken for those of post-mortem introduction.

2. "When the poison has been applied after death, the alteration of the tissues never extends but a small distance beyond the point to which the application was made, so that a *well-defined line of demarkation* is noticed between the healthy and the diseased tissue,—a phenomenon never observed in other cases. The irritation and inflammation resulting from irritant poisons administered during life, although varying in intensity, always extend beyond the point of contact, insensibly decreasing the farther we advance, and never exhibiting a well-defined *line of demarkation.*"

3. "The redness, inflammation, ulceration, and other lesions are much more decided in cases where the poison has been taken during life: hence, if, on examining the body, the rectum or stomach is found covered over with a *large* quantity of one of these poisons, whilst at the same time the lesions are only slightly marked, the presumption would be that the poison had been applied after death."

4. "Irritant poisons introduced into the digestive canal *twenty-four hours after death* do not occasion either redness or inflammation, because the vitality of the capillaries is entirely extinct. If the substances are applied *within one or two hours after death,* they may possibly determine a slight congestion of the mucous membrane; but it would be easy to discover the error."

5. "In cases of this nature it must not be forgotten that poisons are *not rapidly* transmitted by imbibition to distant organs after death, even when the digestive canal contains a large amount of them; and when they do reach these organs, they are found first on their lower surface—that which was the nearest to the poison. It should also be understood

that the poison will not yet have penetrated into the interior of the solid viscera, while it may be detected *on their surface;* so that it can be extracted from a portion taken from the surface of the organ, whilst it would be sought for in vain in its interior. The case is altogether different where the poison has been taken and absorbed during life: then its presence can be demonstrated in any portion of the organ subjected to the examination."

6. "If the body is not examined for several months after death, when the putrefaction of the alimentary canal would not permit an investigation of the pathological changes which might have existed, and more particularly if, after a still longer lapse of time, the whole of the viscera should be mingled in one indistinguishable putrefied mass, it will be impossible for the expert to arrive at any definite conclusion in relation to the mooted question, without an attentive inquiry in relation to the previous symptoms of the deceased, the nature and duration of the sickness, together with all the *moral* circumstances connected with the case—such as the existence of a motive on the part of the accused, the proof of purchase or possession of the poison by him, his *opportunities* for introducing it into the dead body, etc. On the other hand, it may be possible to show that the person who has made the charge against another had the very poison in his own possession,—that he had made a solution of it,—that he was in possession of a syringe or sound, in the interior of which the remains of the poison may possibly still be found, —that he has been seen near the body, turning it over from side to side, etc. Moreover, it may be possible, by a careful examination of the other organs of the body, to prove satisfactorily that death had resulted from natural causes." (Toxicologie, 1852, i. p. 63.)

CHAPTER V.

EVIDENCES OF POISONING.

A KNOWLEDGE of the evidences of poisoning constitutes the chief business of the toxicologist; this it is which enables him to reach a definite conclusion in the cases submitted to his investigation. These evidences comprise, (1) those derived from the *symptoms;* (2) those obtained from the *post-mortem examination;* (3) those afforded by the *chemical analysis;* (4) those derived from *experiments on living animals.* Besides these, there are some collateral proofs, which may be grouped under the name of (5) *moral evidence*, and which at times aid very materially in clearing up a suspicious case. These "evidences" will now be considered in their order.

SECTION I.

EVIDENCES AFFORDED BY THE SYMPTOMS.—DISEASES WHOSE SYMPTOMS RESEMBLE THOSE OF POISONS.

I. *Evidences afforded by the symptoms.*—In any case of sudden death occurring in a person of previous good health, without any *apparent* morbid cause, the suspicion of poisoning is readily awakened in the minds of many persons. These do not consider that sudden death from disease is of frequent occurrence, and they are therefore disposed to assign the cause of such a death—especially if the surrounding circumstances happen to be of a suspicious character — to secret poisoning. Such cases demand the most scrupulous investigation by the legal physician. He should undertake it with a mind perfectly unbiased in any direction. Whilst minutely examining into every detail for the eliciting of the truth, he should be most careful not to express his opinion until the whole matter has been thoroughly sifted,—until every means of research has been exhausted,—lest by so doing he give rise to suspicion against an innocent person, whose character and reputation may thereby be irreparably injured. It is well to understand, at the outset, what is the true value of the *symptoms* exhibited by a person supposed to be poisoned, inasmuch as serious mistakes have resulted from attaching an undue importance to them. We regard these symptoms as constituting *one* important factor in the problem to be determined; but we think that no medico-legal case of poisoning can possibly be established by symptoms alone, for the reason that there are no *characteristic* symptoms of any single poison; if this were possible, and were generally admitted to be so, it is evident that there would be no necessity for a chemical analysis. But no court or jury would be satisfied to rest a capital case upon this one item of evidence. Notwithstanding this, it occasionally happens that an "expert," with a zeal outstripping his knowledge, is betrayed by his ardor into stating that from the symptoms alone he had arrived at the conclusion that poison had been administered. Of course, such a blunder could happen only to some over-anxious individual improvised for the occasion into an "expert:" the practiced toxicologist knows better, and is therefore more reserved in his opinion.

The strong language of Sir R. Christison is very much to the point. "Not many years ago," says this author, "it was the custom to decide questions of poisoning from the symptoms only; . . . it is now laid down by every esteemed author in medical jurisprudence that the symptoms, however exquisitely developed, can never justify an opinion in favor of more than high probability." (On Poisons, 1845, p. 45.) Again he says: "In contrasting the symptoms of poisoning with those of natural disease, no one can hesitate to allow that from them alone a medical jurist can never be entitled to pronounce that poisoning is certain. At the same time, he must not, on that account, neglect them. For, in the first place, they are of great value, as generally giving him the first hints of the cause of mischief, and so leading him to search in time for better evidence. Next, they will often enable him to say that poisoning was possible, probable, or highly probable; which, when the moral evidence is very strong, may be quite enough to decide the case.

Thirdly, although they can never entitle him to say that poisoning was certain, they will sometimes enable him to say, on the contrary, that it was impossible; and to conclude, when the chemical or moral evidence proves that poison was given, the character of the symptoms may be necessary to determine whether it was the cause of death" (*loc. cit.*, p. 55).

It is but fair, however, to the above authority to say that he is of the opinion that, in certain exceptional instances, where the symptoms are of a peculiar and violent character, these may be sufficient to settle the diagnosis. These instances are poisoning by the *corrosive acids, oxalic acid, arsenic*, and *corrosive sublimate* (the two latter under certain conditions). These alleged exceptions will be examined under their appropriate heads.

Orfila (Toxicol., i. p. 10) is very decided in the opinion that a case of poisoning can never be proved by symptoms *alone*. After criticising Christison's assertion that oxalic acid poisoning might be sometimes established by the peculiar symptoms, he shows that this opinion differs materially from that published by the same authority, in conjunction with Dr. Coindet, in the Archives Générales de Médecine, ii. p. 276, in which he states that "even when *all* the symptoms of this poison (oxalic acid) are present, *more certain evidence* will be afforded by the post-mortem lesions, and the chemical analysis." Orfila then proceeds to say: "It will be sufficient to affirm, in order to refute so dangerous an assertion, that there probably does not exist a single case of poisoning occasioned by the substance designated by Dr. Christison, that may not be readily simulated by some disease."

We are now prepared to enter upon the study of these symptoms, which, we are quite ready to admit, constitute a very important link in the chain of evidence in a case of poisoning; and in certain peculiar cases, as *e.g.* the *corrosives*, they go very far towards establishing the proof.

1. *The sudden occurrence of the symptoms in a perfectly healthy person soon after taking food, drink, or medicine.*—It is the property of most poisons, when taken in full doses (as is usually the case when they are criminally administered), to produce their effects very speedily after being swallowed; these cannot be

delayed for any length of time. In some instances the effect is almost immediate, as in the case of prussic acid and nicotine; oxalic acid and strychnia, in large doses, manifest their effects within a few minutes; and arsenic, together with most of the irritant poisons, exhibits its power generally within half an hour. Such is the general rule; but this may be modified by various causes, such as quantity, state, mode of combination, fullness of the stomach, and sleep; the latter may advance or retard the rapidity of action. Besides, if the poison be administered in small and repeated doses, constituting what is termed "slow-poisoning," the effects are altogether different, and may very readily be mistaken for those of some natural disease.

What is chiefly important to be remembered under this head is the fact that many diseases break out just in this manner, suddenly and unexpectedly, in persons previously healthy (or apparently so), and this, too, soon after partaking of a meal. Illustrations are afforded in apoplexy, disease of the heart, perforation of the stomach, cholera, etc.: consequently, too much stress should not be laid upon this one symptom. A suspicion of poisoning is often successfully rebutted by showing that no food, drink, or medicine had been taken for several hours before the symptoms manifested themselves. An illustrative case quoted by most authors is that of the Crown Prince of Sweden, who, while reviewing some troops, suddenly fell from his horse, and died in half an hour afterwards. Suspicion of poisoning was strongly excited, which, however, was satisfactorily negatived by its being shown that the prince had partaken of neither food nor drink for four hours previous to his sickness. His death had really been caused by apoplexy. When, however, symptoms resembling those of poison speedily follow the introduction of food or medicine into the stomach, there may be great room for suspicion; but caution should be observed in drawing inferences, since extraordinary coincidences present themselves. The cases of Sir Theodosius Boughton and M. Fougnies may be cited as good illustrations of actual poisoning. The former of these was poisoned by his brother-in-law, Capt. Donellan, who substituted laurel-water in place of his accustomed medicinal draught: the fact that alarming symptoms came on *two minutes* after swallowing the potion constituted an important part of the evidence. In the case of Fougnies, death took place in *five minutes* after swallowing nicotine, which was forcibly administered to him by his brother-in-law, the Count Bocarmé. As an illustration of the coincidence of a fatal disease suddenly occurring after partaking of food, the case of M. Pralet may be cited. He had dined as usual, in company with his nephew, his heir-at-law; a few hours afterwards, almost immediately after swallowing a glass of wine, he staggered, complained of feeling ill, and became insensible. He subsequently recovered his consciousness partially, but relapsed, with symptoms of paralysis, and died, without convulsions, in about eight hours after the first seizure. On opening the body, great congestion of the brain, together with extravasation of blood, was found; a peculiar odor was also perceived, which was *thought* to resemble that of prussic acid more than anything else! and *faint traces* of the poison were alleged to have been detected. On the strength of this evidence, the nephew was arrested and tried for poisoning his uncle with prussic acid. He was convicted and sentenced; and was saved from execution only by the timely interference of Orfila, who showed clearly that death was solely the result of disease! (Ann. d'Hyg., 1841, ii. 399; and 1843, i. 103 and 474.)

The medical man cannot be too cautious about expressing his opinion in cases of this character. He should never forget that the symptoms before him may really be those of disease. We deem such a person guilty of a flagrant wrong when he gives publicity to the idea of poisoning guided merely by the symptoms alone. *Unless he has first properly analyzed the suspected food or drinks, the vomited matters, and above all the urine*, he has no right to ventilate the suspicion of poison, and thus to implicate a possibly innocent person. If death is not the consequence, and there has been no chemical examination, as above mentioned, there is no possibility of clearing up the case; and, as Dr. Taylor remarks, "No public retraction or apology can ever make amends for the injury which may, in this way, be inflicted on the reputation of another." If death ensues, there is always the opportunity of confirming or refuting the suspicion of poisoning by a careful post-mortem and chemical examination.

Cases are constantly occurring of this false imputation of poisoning, arising from a mistaken view of the *symptoms* by the medical attendant. Dr. Taylor (On Poisons, p. 117) alludes to "certain symptoms following the use of food containing no poison, and yet from taste and smell, as well as the coincidental effects, there will be a strong disposition to charge the crime of poisoning on others." He speaks of many such cases falling under his own observation in which the idea of poison had been taken up, and so persisted in that even the results of a chemical investigation were doubted! "In such cases, the symptoms appear to result from the force of imagination" (*loc. cit.*).

A case of some notoriety (State of Maryland *vs.* Mrs. E. G. Wharton, for attempting to poison Eugene Van Ness, Annapolis, January, 1873) affords a good illustration of the position above taken in regard to the inconclusiveness of "symptoms" when regarded alone. It was alleged that the prisoner had attempted to poison Mr. Van Ness with prussic acid, tartar emetic, and strychnia, on several different occasions; and the suspicion was based solely on the fact that he had exhibited certain symptoms which the "experts" for the State testified were such as indicated, in their opinion, the presence of the above-named poisons. The defense contended that all these "symptoms" were perfectly compatible with ordinary sickness (Van Ness had at times complained of somewhat similar symptoms); and, further, that some of them were *not* compatible with the alleged poisons. Moreover, as the medical attendants who acted as "experts" in the case had neglected to make any chemical examination of the suspected food, or the matters vomited, or the urine, and had administered no antidote, at least for the alleged tartar emetic, or prussic acid, we think there was an entire failure of medical proof of the alleged poisoning. The result was a disagreement of the jury; and before the second trial came on, the State virtually abandoned the case. (See Review of the Medical Testimony in this trial, by Dr. H. C. Wood, in N. Y. Medical Record, 1873.)

2. The additional fact that *several persons, partaking of the same food or drinks, all exhibit the same symptoms.*—This materially strengthens the evidence from the symptoms, since it is not likely that several persons should be attacked by a similar disease, just at the same period of time. Nevertheless, to show how such a case may be possible, Dr. Taylor (On Poisons, p. 119) mentions the case of a family consisting of four persons who sat down to dinner in London, all apparently in good health. Soon after the meal three of these were seized with violent symptoms of irritant poisoning. Two of the persons died. The son, who was the only member of the family who did not suffer, and who was known to be hostile to his parents, was arrested on suspicion of having poisoned them; but a strict investigation of the case subsequently proved that the deaths had been occasioned by malignant cholera, which was prevailing in London at the time.

It is hence evident that even a simultaneous occurrence of suspicious symptoms merely furnishes a *presumption* in favor of poisoning,—to be supported or rebutted by other circumstances. If, in the above case, or in others similar, a chemical analysis had detected poison in the food or vomited matters, or in the excreta of the sufferers, of course there could be no hesitation in referring the symptoms to poison; and yet it can be seen how, even in such an extreme case, there might possibly be a coincident disease. If such a contingency should ever happen, it will become the duty of the medical jurist to apply those rules of discrimination which are resorted to in cases of violent death from injuries.

On the other hand, the mere want of this coincidence of symptoms among a number of persons who have partaken of the same meal, is no proof of the absence of poison; for it may readily happen that the poison has been introduced into only a certain portion of the food,—as in the gravy and not in the meat, in the crust of a pie and not in its contents, or in the sauce and not in the pudding, or *vice versa.* Hence it will turn out that only those who have partaken of the one special article of food or drink manifest the symptoms of poisoning.

It may be well here to remark that obscure symptoms of poisoning may occur simultaneously in several members of a family, which may give rise to unfounded suspicions against an innocent individual. These may generally be traced to an accidental introduction through the food or drink, as from copper or lead, through a want of proper care and cleanliness. Finally, it should not be forgotten that symptoms of irritant poisoning are occasionally manifested simultaneously in a number of persons who have eaten food rendered unwholesome by disease or decay. (See *post.*)

3. *The rapid course of the symptoms towards a fatal termination.*—Although this feature is generally regarded as indicating the presence of a poison, it is not of much practical value as a means of diagnosis; since many of the most active poisons not unfrequently prove fatal after a protracted interval, whilst many diseases run their course very rapidly.

From what has been said in relation to diagnosticating a case of suspected poisoning, by the *symptoms*, it will be apparent that the real difficulty consists in discriminating between these, and the symptoms exhibited by disease. It will therefore be proper briefly to consider the disorders whose symptoms most resemble those of poisoning.

The diseases most likely to simulate *irritant* poisoning are cholera, inflammation of the stomach and bowels, ulceration and perforation of the stomach, peritonitis, ilius, and strangulated hernia. Those that may simulate *neurotic* poisoning are apoplexy, epilepsy, inflammation of the brain, tetanus, and diseases of the heart. These will be briefly considered.

Cholera.—(*a*) Common cholera morbus resembles in many respects, in symptoms, several of the irritant poisons, particularly arsenic. Thus, it often comes on within an hour after eating indigestible food; there are violent vomiting and purging, with cramps; a cold skin, feeble pulse, thirst, and depression of strength. Mistakes have frequently been made in confounding this disease with arsenical poisoning. The following are the most distinctive points: Cholera morbus is usually dependent on some irregularity of diet, and appears chiefly at the latter end of summer; poisoning by arsenic may occur, of course, at any season. The dejections of cholera morbus are always tinged with bile, and never with blood; in poisoning, they very often contain blood and mucus. In cholera, there is wanting the sense of burning and constriction of the throat, which are prominent symptoms in arsenic-poisoning. Besides, there are often certain special and occasional symptoms present in poisoning from arsenic which are wanting in cholera morbus, as the redness of the conjunctiva, the eczematous eruption, and the paralysis. The only *certain* means of diagnosis in a non-fatal case is a chemical examination of the food, the matters vomited, and especially the urine (*vide post*, ARSENIC).

(*b*) The symptoms of *Asiatic cholera* more strongly resemble those of tartar emetic than of arsenic. The copious watery discharges from the stomach and bowels, the profuse cold perspiration, the cramps, the cold and sometimes livid skin, the feeble, fluttering pulse, the extreme prostration—occur in each case. A microscopic examination of the discharges in cholera, would doubtless lead to a correct diagnosis.

Inflammation of the stomach and bowels (*Gastro-enteritis*).—As the irritant poisons spend their force chiefly upon the lining membrane of the alimentary canal, it is not surprising that their most prominent symptoms—vomiting, purging, and abdominal pain—should be mistaken for the ordinary signs of gastro-enteritis. The following are the points of divergence in their symptoms: Idiopathic gastritis is a very rare disease; when gastritis does occur, therefore, it may generally be traced to some direct irritating cause, such as poison. The fact of the violent symptoms coming on very soon after a meal, would excite suspicion, although, of course, this would not be conclusive. Again, there is, in cases of poisoning, usually an absence of the fever which accompanies the disease.

In *peritonitis*, the constipation would serve as a distinctive mark to prevent its being mistaken for irritant poisoning. *Strangulation of the bowels*, *intussusception*, and *strangulated hernia* have sometimes given rise to suspicion of irritant poisoning, chiefly from the sudden accession of pain, and the vomiting; but a careful examination of all the circumstances of the case, and especially the post-mortem investigation in a fatal case, will reveal its true nature.

Among the less frequent diseases may be mentioned *rupture of the stomach and duodenum*,—instances of which are mentioned by Christison as having occurred under circumstances which might easily have suggested irritant poisoning; *rupture of the biliary ducts; rupture of the uterus* (of which a striking case is given by Christison, and which became the subject of judicial investigation as a case of poisoning); and the *effects of drinking cold water*. In the above cases, the post-mortem examination alone could clear up the diagnosis.

Diseases simulating neurotic poisoning. *Apoplexy.*—The symptoms of this disease so closely simulate those of some of the narcotic poisons, especially opium, that it is often impossible to distinguish between them by these symptoms alone. The following characters are usually given as diagnostic marks: Apoplexy is comparatively of rare occurrence before the age of thirty years; it is often preceded by certain premonitory signs; its symptoms may not come on for several hours after food or drink has been taken,—which would negative the idea of poisoning (see *ante*, p. 47); the coma frequently comes on suddenly, whilst the narcotism from opium is gradual. The pupils are dilated in apoplexy, but contracted, as a rule, in opium-poisoning.

To the above general symptoms connected with apoplexy there are numerous exceptions: thus, apoplexy does occasionally happen in the young, and even in infants; in some instances it comes on suddenly, and soon after eating; occasionally the pupils are *contracted* in apoplexy (see Dr. Wilks' cases of apoplexy of the pons Varolii, Med. Times and Gaz., 1863); and in the latter stages of opium-poisoning they are frequently dilated. In fatal cases of opium-poisoning the vessels of the brain and dura mater are generally gorged with blood; sometimes there is an effusion of serum into the ventricles and beneath the membranes; but no extravasation of blood into *the substance* of the brain, as is usual in apoplexy.

Epilepsy.—Some of the symptoms of this disorder partially resemble those of prussic acid poisoning; but the history of the case will usually be sufficient to clear up all doubts as to its real character. The first epileptic paroxysm is rarely, if ever, fatal.

Tetanus.—This disease is very seldom of spontaneous origin, or idiopathic; it is almost always the result of an injury inflicted upon tendons, nerves, aponeuroses, or fasciæ; and frequently it can be traced to very slight injuries of these parts of the body. Tetanic convulsions are also a characteristic result of poisoning by nux vomica and strychnia. They occasionally also manifest themselves in that protean disorder, hysteria; and exceptionally, in poisoning from arsenic, antimony, morphia, and some other poisons. The following are the diagnostic signs distinguishing ordinary tetanus from that produced by strychnia: in ordinary tetanus, the spasm comes on gradually; the rigidity commences in the jaws, and then advances progressively to the muscles of the neck, trunk, and extremities. According to Mr. Colles (Lect. on Surg., i. p. 72), the muscles of the fingers are the last and least affected. This rigidity continues more or less uninterruptedly throughout the whole course of the disease. In tetanus from strychnia, the spasm comes on suddenly, after a shivering, attacking the whole body and limbs simultaneously, and affecting the muscles of the jaw last. The paroxysm lasts a few minutes, when a complete intermission occurs, during which there is a perfect relaxation of all the muscles. The *duration* of the symptoms is also a distinctive mark: whilst the rigidity of the disease may continue for days, the convulsions of strychnia rarely last over two hours; often they terminate in death in less than half an hour. In the convulsions of strychnia, the intellect is unaffected, even in the paroxysms; whilst in tetanoid hysteria there is often loss of consciousness, and convulsive motions of the limbs alternate between stiffness or rigidity; moreover, whilst in tetanus from strychnia the paroxysms are very apt to terminate fatally, in hysteria they never do. Finally, the absence of any wound, burn, or marks of an injury or of exposure to cold would negative the idea of disease, and rather point to strychnia as the cause.

Distension and rupture of the stomach may result in sudden death, with symptoms of apoplexy; and, as these usually come on after hearty eating, a suspicion of poisoning may arise. A proper examination of the body after death will alone reveal the nature of the case.

SECTION II.

EVIDENCES FROM POST-MORTEM APPEARANCES. — LESIONS COMMON TO DISEASE AND POISONS.

II. *Evidences derived from post-mortem appearances.* — The evidence obtained from this source, like that derived from the symptoms, can never be absolutely conclusive: it can merely be more or less suggestive. Only a few poisonous substances make such decided impressions upon the living tissues as to warrant even a very strong presumption of their presence: these are the caustic acids and alkalies, which usually produce very decided pathological changes—such as erosion, perforation of the stomach and bowels, alteration of color, softening of tissue, etc. Yet there have been fatal cases of poisoning from these substances, with an absence of their usual corrosive effects after death.

According to Prof. Wormley (Micro-Chem. of Poisons, p. 45), "some poisons leave no appreciable morbid changes in the dead body; and of those that usually do, the appearances are subject to great variety, and in many instances are similar to the effects of ordinary disease, or even the results of cadaveric change."

Dr. Taylor remarks (On Poisons, p. 154) that "any evidence derivable from appearances in the body of a person poisoned will be imperfect, unless we are able to distinguish them from those analogous changes often met with as the results of ordinary disease."

It was formerly supposed that in cases of death from poisoning, the *external appearance* of the body presented a peculiar character, such as an unusual degree of lividity and a tendency to rapid putrefaction. This idea is erroneous, so far as relates to the great mass of cases. The exception seems to be in poisoning by sulphuretted hydrogen, in which case putrefaction does occur more rapidly than in death from other poisons; on the other hand, arsenic, alcohol, and sulphuric acid appear to exert an anti-putrescent influence upon the body.

Under this head, we may mention certain external marks upon the body — dead or living — which would be very suggestive of particular poisons, as, for example, the peculiar stains of the mineral acids on the lips, cheeks, tongue, fauces, etc., and sometimes even on the *dress* of the person. Sometimes the odor of certain poisons may be detected even before opening the body,—*e.g.* that of prussic acid, opium, alcohol, or nicotine. The open, staring appearance of the eye, together with livid spots about the face, neck, and hands, is suggestive of poisoning with prussic acid; a very long-continued rigidity of the body after death usually attends cases of strychnia-poisoning.

In relation to the proofs of poisoning presented by an *internal* inspection of the body, valuable information may sometimes be gained from the peculiar *odor* exhaled. Thus, frequently, though not always, on opening the body of a person who has recently died from prussic acid poisoning, a very strong odor of that substance can be perceived. The same is sometimes the case where death has resulted from opium, or its liquid preparations; and in *phosphorus*-poisoning, the alliaceous odor and the white fumes of phosphorous acid are strongly characteristic of that poison. In poisoning from nicotine, the peculiar penetrating odor of this substance may often be recognized in the dead body; and in cases of death from sulphuretted hydrogen, the presence of this poisonous gas is detected in a similar manner. So, likewise, in some cases of poisoning with corrosive sublimate, there is found, after death, a peculiar, slate-colored deposit on the lining membrane of the stomach, consisting of reduced mercury in a finely-divided state; and at times, as the result of arsenical poisoning, there may be seen white patches, composed of arsenious acid, firmly attached to the highly-inflamed mucous coat of the stomach: such patches of inflammation are very characteristic of this poison. Again, the remains of various poisons can be recognized after death, *in the stomach* *and bowels*, by close inspection either by the naked eye, or with the aid of the microscope. Thus, cantharides, when swallowed in powder, Scheele's green, and orpiment, nux vomica, savin, and various other vegetable leaves and seeds, present characteristic marks, botanical and otherwise, by which they can be identified, after death.

But there are certain alterations of the tissues and organs themselves — true pathological lesions — resulting from the direct action of poisons, which it is important to understand. As a rule, the *irritant* poisons leave behind them very decided evidences of their action on the gastro-intestinal mucous membrane, such as redness more or less diffused and intense, thickening, softening, ulceration, perforation, and gangrene. So, likewise, the *neurotic* poisons leave their impress upon the nervous centres, in the form of congestion of the vessels of the brain and spinal marrow, effusion of serum (rarely of blood) into the ventricles of the brain, and beneath the membranes; sometimes congestion of the lungs, general fulness of the venous system, and of the right side of the heart, etc.

Considerable importance attaches to the negative evidence from post-mortem inspection. Thus, in a case of alleged poisoning by sulphuric or nitric acid, the absence of all marks of corrosion in the mouth, gullet, and intestinal canal would negative the charge; but while the absence of all traces of inflammation of the mucous membrane of the stomach and bowels would render the fact of poisoning by arsenic, corrosive sublimate, and other irritants highly improbable, it would not necessarily invalidate it, since, as we shall learn hereafter, death may occur from these powerful irritants without leaving behind any pathological lesion. The negative evidence of absence of congestion of the brain, in cases of narcotic poisoning, is weaker.

It must also be remarked that post-mortem appearances similar to those above mentioned, even though confirmed by the chemical detection of the poison in the stomach, do not furnish absolute proof that death was *caused* by poison; for this might, after all, be due to some cause anticipating its fatal action. Such a coincidence could be determined only by a careful post-mortem examination. The detection of the *absorbed* poison in the viscera would go still farther to establish the proof of poisoning. On the other hand, a dead body may bear upon it very positive evidence of violence, such as bruises, stabs, and other wounds, sufficient to account for death, and yet on inspection this may be found to have been actually caused by poison.

It has already been stated that there are many post-mortem appearances common to poison and disease,—just as is the case with *symptoms:* it is important to have a clear understanding of these, so as not to confound them, in making an inspection.

Redness.—This is one of the most common of all the effects of an irritant poison; but it is likewise a very frequent consequence of *disease;* and, according to the highest authorities, it is often the result of post-mortem changes, independently of any antecedent disease. The researches of Drs. Yellowly and Andral have put this matter beyond a doubt: hence *mere redness* of the stomach cannot be accepted as any proof of poisoning. In ordinary gastro-enteritis, where the mucous membrane of the stomach and bowels is highly inflamed, redness is, of course, a prominent sign; and inasmuch as the symptoms before death are very similar to those occasioned by arsenic, or some other irritant, it would be impossible to make out the diagnosis clearly without a chemical analysis; and the result even of this would not necessarily be final or positive; for supposing only a minute quantity of arsenic to be discovered—not in the *contents* of the stomach, but in its tissues,—and in the liver and other viscera of the body, this circumstance *alone* would not be positive proof of death from poison. For it might so happen that the individual had been taking arsenic *medicinally*, in small doses, for weeks or months previously, and then have died suddenly from an acute attack of gastro-enteritis, under suspicious circumstances; and after death, the suspicion would *appear* to be sustained by the chemical discovery of the absorbed poison in the viscera. We have met, in our own experience, with just such a case, in which the success of the defense consisted in showing that the absorbed arsenic detected in the liver of the deceased could be satisfactorily accounted for by her having taken this substance medicinally for a considerable time prior to her death: the prisoner was acquitted. If, however, in a doubtful case of the above character, where the symptoms and post-mortem lesions were consistent with poisoning, the poison had been found *in the contents* of the stomach, and also in the absorbed state, in the liver, etc., such a discovery would furnish positive proof of the crime, unless it could be shown that the poison had been introduced into the stomach after death.

It is proper to state, on the other hand, that when internal redness of the stomach is really due to arsenic-poisoning, this condition may continue, and be recognized many months after death, in consequence, probably, of the antiseptic influence of the poison upon the tissues.

Ulceration.—This result is rare as a consequence of acute irritant poisoning; but it occasionally occurs as one of the sequences of chronic or slow poisoning by arsenic. It is believed to be more commonly the result of disease than of poisoning. It is a very insidious disorder, often lasting for a considerable time without causing much inconvenience to the patient, except occasional nausea and vomiting, and loss of appetite. Dr. Taylor draws attention to the fact that ulceration from poisoning has never been known to occur until *after* the appearance of symptoms indicative of irritant poisoning: this fact would aid us in making the diagnosis. In ulceration from disease, the mucous membrane is commonly only reddened immediately around the ulcer; in ulceration from poisoning, the redness is generally diffused throughout the membrane, and extends into the small intestines. The clinical history of the case will aid in clearing it up.

Ulceration must not be confounded with *corrosion:* the former is a vital process,—the parts being removed by absorption; the latter is the result of chemical action, at once destroying their vitality. It is also a very rapid process: the former is slow. The complete and thorough destruction of tissue produced by the mineral acids, can hardly fail to disclose the real cause of death. There is no disease which could produce such well-marked appearances *in so short a time.*

Softening.—The mucous lining of the stomach and bowels may undergo this change, both as the result of poisoning and of disease; also, according to good authority, from the post-mortem solvent action of the gastric juice. In some cases, the coats of the stomach become thickened and hardened under the effects of arsenic and other irritants. We are not warranted, *from softening alone*, to infer the existence of an irritant poison. The absence of the peculiar symptoms and of other appearances indicative of poison, together with a failure to discover it by a chemical analysis, would be quite sufficient to disprove such a charge.

Perforation.—When this is the result of poisoning, it is almost exclusively due to the *corrosives*, especially the mineral acids. In such cases, the aperture is large and ragged; the edges are soft and friable; the poison escapes into the abdomen, and can there readily be detected. In perforation from disease (ulceration), the aperture is small; and it often comes on very suddenly after a meal, thus giving rise to a suspicion of poisoning. It is usually fatal, death being ascribed to the consequent peritonitis.

The following are the diagnostic points: in poisoning, it is preceded by very severe symptoms; in disease, the preceding symptoms are generally mild, and often resemble those of dyspepsia; the attack, however, comes on very suddenly after eating; vomiting is usually absent, or else is very slight; and instead of purging there is constipation. These, together with the difference in the appearance of the aperture, and the result of the chemical examination, will serve to show the true nature of the case.

Cases of sudden *perforation of the intestines* from disease are reported, which gave rise to a suspicion of poisoning, the true nature of which was ascertained only by a post-mortem examination. There can be no doubt that the intestines have occasionally been perforated by *worms* infesting the bowels. A case is reported by Flandin, where this was made a ground of defense (Taylor, On Poisons, p. 165).

SECTION III.

INSPECTION OF THE BODY.—COLLECTION AND PRESERVATION OF THE SUSPECTED MATERIALS.

Inspection of the body.—This is the appropriate place to offer some suggestions in reference to the proper mode of inspecting a dead body with a view to determine the question whether the death was caused by poison. The duty of the expert may have reference, (1) to the examination of a body recently dead, and (2) to the exhumation and examination of a body dead for a long time.

In reference to the former, the expert should ascertain, if possible, the exact time of death, and compare this with the first appearance of the symptoms, so as to determine how long the person survived after being attacked. He should notice the attitude and position of the body, as indicating convulsion—tetanic or otherwise—during life; the state of the countenance, whether livid or pallid, ecchymosed or not; the condition of the dress, whether exhibiting marks or spots of the mineral acids, or of vomited matters; likewise the condition of all surrounding objects, whether stained with vomited matters, blood, etc. He should carefully collect any vomited matters, and remove any suspicious stains from the floor, articles of furniture, etc., by scraping, or otherwise. He should collect, and carefully lay aside, all suspicious papers, boxes, cups, bottles, etc., for further examination.

When a considerable length of time has elapsed since the death occurred, and for some reason the suspicion of poisoning has been aroused, a judicial order is obtained, and it becomes necessary to disinter the body. The expert should always be present at the exhumation of the body, and give his attention to the minutest details connected with this procedure. He should take notes of the mode of burial; the nature of the soil; the condition of the coffin, whether sound or decayed, and the state of the clothing of the body. If the body be in good preservation, and the coffin sound, the former should be lifted out, and laid upon a table near by, suitable for making the autopsy; but if, from the long period of interment, the body be much putrefied, and the coffin much decayed, it will be proper to collect some of the débris adherent, as well as some of the adherent earth, and likewise some of the soil taken from another part of the cemetery, for a comparative analysis.

The rules governing the autopsy are the same as those which regulate other judicial post-mortem examinations. The first thing to be noticed is the condition of the body—whether well preserved or not; and whether it has been embalmed. One general rule should regulate every such investigation, namely, that it should be so thorough and exhaustive as to leave no single organ of the body unexamined. We cannot be too emphatic upon this point. Tardieu (Sur l'Empoisonnement, p. 57) uses very decided language on the subject: "Most unquestionably this post-mortem examination should be complete, without omitting a single organ, so as to overlook no lesion whatsoever, and no cause of either natural or accidental death."

The evils resulting from a careless or superficial autopsy, in such a case, will be apparent on a moment's reflection. Cases of poisoning constantly occur, where the ends of justice are imperiled or defeated by just such an imperfect post-mortem examination. Suppose a case of alleged poisoning by opium: the *symptoms* strongly resemble those of apoplexy; the autopsy is conducted in this careless, slovenly manner; the brain not minutely inspected for disease; the spinal cord altogether omitted; the kidneys and other organs neglected. The incompetent "expert," however, ventures upon the witness-stand, and presumes, even under the solemn surroundings of a case of life or death, to give his opinion that death was caused by opium-poisoning, *because* he had discovered some congestion of the brain, with, perhaps, some effusion! no microscopic examination of the minute cerebral vessels, or of the heart; no inspection of the kidneys for granular disease to indicate uræmic poisoning; and, most probably, no examination of the urine to confirm or rebut this suspicion! It is positively alarming to contemplate the danger to which, on the one hand, innocent persons, wrongfully accused, may be exposed, through such ignorant and careless work, or, on the other hand, how, by creating doubts in the minds of the jury, a really guilty person may fail to be convicted.

In cases of suspected poisoning, it is generally recommended to place a double ligature at each end of the stomach, and to cut between them, so as to preserve this organ with its contents entire for future examination. Tardieu, however (*loc. cit.*, p. 58), strongly advises to examine the interior of the stomach and intestines on the spot, before any further putrefactive change occurs, which is very apt to be the case. For this purpose, he recommends removing the stomach carefully from the body, without tying it, and then emptying the contents into a proper vessel: the intestine is to be emptied by placing the upper end in the vessel, and then detaching it from the mesentery, gradually pushing the contents into the vessel. After this, it will be easy, by cutting open the stomach and intestines, to examine their inner coating. Whichever mode the expert may prefer to adopt, he should be provided, in advance, with several new and clean wide-mouthed glass or stone jars, of the capacity of half a gallon, fitted with sound *new* corks or glass stoppers. One of these should contain only the stomach and the intestines; or, if only a portion, the upper third, together with the cæcum and rectum. None of the other viscera should be placed in the same jar, lest, by imbibition, these might become contaminated with the poison in the stomach, and so lead to a wrong inference in relation to *absorbed* poison (see *supra*, p. 40). The second jar may contain portions of the liver and lungs, spleen, heart, kidney, and the urinary bladder. It is very important to avoid putting any antiseptic liquid into these jars. Even pure alcohol, or chloroform, allowed by some authorities, is, at times, objectionable, as, for example, where phosphorus is to be looked for. The jars should then be securely corked, avoiding the use of sealing-wax (which contains some mineral ingredient), but fastened by tying over the corks a piece of moistened bladder, or parchment; after which they are to be properly sealed up, and kept under lock and key, so as to prevent the possibility of their being injured, or in any way being tampered with.

The importance of receiving the stomach, etc., into a perfectly *clean* vessel may be inferred from the fact, that the showing that this vessel was not perfectly clean, at the trial, would be sufficient to destroy all the chemical testimony, however conclusive this may be as to the discovery of poison. This is well illustrated in a case communicated to the author by Prof. R. Bridges, of Philadelphia, which occurred to himself. In a case of suspected arsenic-poisoning, the stomach, etc., were carelessly thrown into an old tin can that had formerly contained zinc paint, before being sent to the analyst. He discovered *zinc* in the viscera, and was at a loss to account for its presence, until he ascertained the above fact.

For a similar reason, the jars, with their contents, should be guarded with scrupulous care, lest it become impossible to vouch for their *identity* at the approaching trial. A great point with the defense at such a trial, is to disprove, if possible, the identity of the materials operated upon by the chemist. A singular case of this character was tried at Leesburg, Va., in 1872 (State of Virginia *vs.* Mrs. E. E. Lloyd), where a woman was accused of poisoning her children with arsenic. It appeared in evidence, that the person who had been intrusted to carry the jar containing the stomach to the chemist, in a distant city, had died in the interim before the trial; and there was no one who could swear to the identity of the stomach operated on by the chemist, who declared that he had extracted from the stomach several grains of arsenic! The court very properly ruled out the whole of the chemical evidence in relation to the stomach, just for the loss of this one link in the chain.

The *contents* of the stomach should be collected in a clean, graduated vessel, and their *quantity*, *color*, *odor*, *consistence*, etc., carefully noted. The condition of the rectum, and of the genital organs of the female, should be inspected; the presence of hardened fæces in the *rectum* would show that purging had not existed shortly before death,—a circumstance which of itself would go far to disprove that death had resulted from an irritant metallic poison, as arsenic, corrosive sublimate, or tartar emetic. It may be well also to remember that arsenic has been introduced into the vagina with a murderous intent.

One other point should not be forgotten, namely, to examine all the important organs for *marks of natural disease*, and to note down any unusual pathological conditions, even though, at the time, these may not seem to bear on the question of poisoning.

In the subsequent examination of the stomach and bowels, after noticing any odor, as indicating the presence of alcohol, phosphorus, prussic acid, opium, etc. (slightly warming the organic matter, if necessary, for this purpose), each portion of the intestinal canal should be in turn spread out upon a clean sheet of window-glass, with the internal surface outwards. The entire surface should then be carefully examined, first with the naked eye, and afterwards with a lens; every abnormal appearance should be noted and described; all suspicious particles or powder should be collected and examined; such portions as present the most marked appearances may then be removed, and spread out upon a glass slide and placed under the microscope. By this means, very satisfactory proof may often be obtained of the presence of certain kinds of food, of certain poisonous vegetable seeds, leaves, chlorophyl, woody fibre, the characteristic granules of the different varieties of fecula, the spores of mushrooms, etc. A striking illustration of the importance of such a minute examination is mentioned by Tardieu (Sur l'Empoisonnement, p. 68). A child twelve years of age died at school, after ten hours of acute suffering, on the day on which its stepmother had brought it several good things to eat. Among the contents of the stomach were discovered some fragments of bread, which, when examined by the microscope, were found covered with a fungous growth, showing that the bread was mouldy. This fact was noted down, but no importance was attached to it till, at the trial, one of the witnesses, a servant of the stepmother, stated that her mistress was in the habit of carrying to the child slices of bread and jam; but that on the day of the death she said that she could not take it, because the bread was mouldy, and had been so for several days.

In conclusion, a judicial exhumation and examination of a body suspected to have been poisoned should never be made except in the presence of the properly qualified officer—the coroner, and always in the presence of some representative of the prisoner, and of the defense; otherwise, the matter assumes very much an *ex parte* character, and the State's expert who conducts it must necessarily be exposed to an unpleasant suspicion of not dealing fairly with the material examined. At any rate, we think it safest and best to avoid all such imputations, by having an expert for the defense present at the examination. An opposite course is calculated to create an unfounded suspicion against even honorable men, who may unwittingly lend themselves to it.

SECTION IV.

EVIDENCE FROM CHEMICAL ANALYSIS. — CAUSES OF FAILURE IN THE CHEMICAL ANALYSIS.—OBJECTS OF THE CHEMICAL ANALYSIS.—PRECAUTIONS TO BE OBSERVED.—ACCURACY OF THE ANALYSIS.—IMPURITIES IN REAGENTS.

III. The actual detection of the poison by means of chemical analysis is generally regarded by the popular mind as affording the most satisfactory and positive evidence of poisoning. Indeed, the idea has been very prevalent that the charge of poisoning can never be made out, without the actual production of the poison as the *corpus delicti.* This is, however, an error. All that the law requires is satisfactory proof that the person has died from poison. *It does not prescribe the means by which this proof is to be arrived at.* The question then is—can satisfactory proof be established *without* the chemical detection of the poison ? The reply to this inquiry is, that it unquestionably can be, in certain cases. There are certain poisons that cannot, at present, be detected by chemical analysis; such are some of those derived from the vegetable and animal kingdoms, as the œnanthe crocata, the laburnum, the poisonous fungi, the woorara, poisonous cheese and meat, the poison of rabies, of glanders, of snakes, etc.; yet all these can produce most violent, and often fatal, effects. It would be, as Dr. Taylor well remarks, a most dangerous and fallacious doctrine to affirm that no case of poisoning can be proved without the actual detection of the alleged poison by chemistry. If this were true, many notorious murderers would be allowed to escape the hands of justice. The truth is, that the fact of poisoning can often be made out by physiological and pathological evidence, conjoined with strong moral proof, without the additional aid of chemistry. In many celebrated criminal cases, conviction has ensued in the absence of all *chemical* proof. We need only mention, among many others, the cases of Donellan, Castaing, and Palmer as illustrations. In none of these instances was the poison found in the body of the deceased, yet in all the "satisfactory proof," which the law requires, was obtained from other sources, and conviction followed.

It becomes, then, a matter of extreme importance to ascertain precisely the position which the *chemical proof* occupies in the chain of evidence required to establish the charge of poisoning. We say, then, that if the other factors of evidence —the symptoms, the pathological appearances, the poisonous effects of the suspected material on living animals, and the moral proofs—all coincide, the chemical analysis is not required to substantiate the charge. Nor is this doctrine opposed to what was before said in reference to the insufficiency of proof, either from the symptoms alone, or from the post-mortem signs alone, or even from both combined; inasmuch as these, unless supported by the strongest *moral* proof, such as purchase and possession of the poison, motive, conduct before and after the victim's death, etc., can merely establish a probability of poisoning. "When, however, the other branches of evidence fail, or are imperfect, if the chemical analysis is unsatisfactory, an acquittal must follow." (Taylor, On Poisons, p. 172.) But on the other hand, even if, under these circumstances, the analysis distinctly reveals the presence of poison, it simply declares the *fact* of its presence: it does not necessarily prove that it was the cause of death. Indeed, in the entire absence of the usual symptoms, pathological changes, and moral proofs, the chemical detection of a poison may rather justify the suspicion that this had been secretly introduced into the body after death, for a sinister purpose. (See *ante*, p. 40.)

Whilst, then, we do not hesitate to admit that there are cases of poisoning which can be "satisfactorily proved," even in the absence of all chemical evidence, we would not be understood as in any way disparaging or underrating the importance of a thorough chemical analysis. When this demonstrates the presence of a poison, it exhibits a positive and sufficient means of death, with the proviso just mentioned, that the poison could not have been introduced into the body after death. It is usually considered the most satisfactory evidence of the crime, by both court and jury.

Orfila evidently lays great stress upon the *chemical analysis* as an important link in the chain of evidence. He says,—speaking of the respective value of symptoms, morbid lesions, and the chemical analysis in a case of poisoning, and assigning a paramount importance to the latter,—"Suppose twenty persons to be poisoned by arsenic: in every one of them the presence of the poison is proven by the analysis, whilst the symptoms experienced, and the post-mortem appearances presented, differ widely in their character. In one, the sickness resembles Asiatic cholera; another, several hours after swallowing the poison, is suddenly attacked by syncope, without any precursory symptom, and dies; later, some will be found affected with pustules on the skin, delirium, faintings, articular pains, abundant and repeated vomitings, etc., while others exhibit only a few of these symptoms, or else all of them in a very slight degree. And as to the *lesions*, in one case we will find ecchymoses in the alimentary canal, with several ulcers, and possibly perforation; in another, a bright, uniform, and extended redness; in still another, a simple injection of the membrane; and in some others even this may be wanting." His conclusion is, that the diagnosis cannot be made out by the symptoms and morbid lesions *combined*, without the aid of the chemical analysis (*loc. cit.*, p. 11).

The great value of chemical evidence is seen in the detection of poisons in bodies many months, or even years, after death, when all appearances are destroyed by putrefaction, and also in the excreta (the urine particularly) of the living. In the absence, or failure, of the chemical evidence, in an alleged case of poisoning, we think that the proofs derived from the other sources should be so positive and unequivocal as not to admit of the shadow of a doubt.

Causes of failure in the chemical analysis.—These are as follows: 1. *The nature of the poison itself:* no chemical test may have yet been discovered for it. As already remarked, this is especially true of certain vegetable and animal substances of a poisonous nature. As a rule, the *inorganic* poisons admit of comparatively easy detection, in the hands of an expert chemist. Some poisons are of an exceedingly volatile nature: they rapidly disappear from the body, by evaporation. This is the case with prussic acid, alcohol, nicotine, chloroform, etc.: so that a chemical examination made a few hours after death, may entirely fail to discover a trace of them.

2. *Loss by vomiting and purging.*—It is evident that even a large dose of poison may be entirely expelled from the stomach and bowels by vomiting and purging, provided these occur soon after swallowing it. Numerous cases are on record where, from this cause, there was an entire failure to discover the poison after death, although this took place with the usual rapidity. Generally, however, when the poison has been taken in the solid state, as in powder, more or less of it remains closely adherent to the mucous lining of the stomach, where it can be easily identified. If the vomiting and purging have not been excessive, and life has not been prolonged over two or three days, a portion, at least, of an *irritant* poison ought to be found in the stomach. The exact period when the whole of a poison disappears from the stomach by vomiting is subject to much variation. Christison mentions two cases of poisoning by arsenic, which proved fatal in five hours, after much vomiting; in one of which no poison could be detected, and in the other only a fifteenth of a grain could be found. In two other instances of like poisoning, in which death ensued in eight hours after swallowing one and two ounces respectively, not a trace of the poison was discovered in the stomach. On the other hand, Orfila mentions a case in which, after incessant vomiting for two entire days, arsenic was detected in the contents of the

stomach. Prof. Wormley (Micro-Chem. of Poisons, p. 37) discovered forty-two grains of arsenic in the stomach of a person who had been vomiting almost incessantly for thirty-two hours.

3. *Loss by absorption and elimination.*—Attention has already been directed to the rapidity with which poisonous substances are removed from the stomach by absorption (*ante*, p. 26). Hence it often happens that, after death from poisoning, even though there may have been no vomiting, the analyst may not be able to discover a trace of the poison in the stomach. This is especially apt to be the case where the dose was not excessive, and where death did not ensue very rapidly. In the year 1861 I examined the stomach and intestines of a woman who survived six hours after swallowing nearly six grains of strychnia. The most careful and repeated analyses by Stas' process failed to reveal a trace of the poison, either by the color-test, or by the bitter taste of the extract. Sir R. Christison failed to detect morphia in the stomach of a person poisoned by taking two ounces of laudanum, although death occurred in five hours (On Poisons, p. 697).

The elimination of the absorbed poison from the different organs of the body, as we have already seen, commences almost immediately after its ingestion; but the time required for the *complete* elimination varies extremely, both for different poisons, and for the same poison under different circumstances. Thus, according to Orfila, the period for the total expulsion of *arsenic* from the human body by elimination is about fifteen days; nevertheless, arsenic has been found in the urine as late as the twenty-first day after the cessation of its administration. From this it follows, that, in case of death from arsenic, if the deceased survived fifteen or sixteen days, there would be no probability of discovering a trace of the poison, either in the stomach, or in any of the organs. Doubtless, in numerous cases, the period of elimination is much shorter than the one just mentioned, as, for example, where the dose has been only just large enough to cause death; but in the great majority of cases of poisoning, the doses are excessive, the symptoms violent, and the death rapid: so that there is usually little difficulty for the chemist to detect the poison, both in the contents of the stomach and in the organs. This is particularly true of the *mineral* poisons; vegetable poisons (alkaloids), although undoubtedly absorbed into the blood, like the others, have very rarely been detected in the organs.

4. *Decomposition of the poison in the blood, or during its elimination from the system.*—It would appear from the difficulty that attends the detection of certain poisons after death,—more especially those of the organic kingdom,—that they probably undergo some chemical alteration in the living body, which renders them insusceptible to the action of the usual reagents. This seems particularly true in the case of opium, and it has also been suggested as belonging to strychnia. In a former chapter (p. 32) proofs were adduced of the fact that many substances do undergo decomposition while passing through the circulation. The non-detection of most of the organic poisons in the tissues and organs of the bodies of persons killed by these substances, furnishes a very plausible argument in favor of their chemical decomposition in the blood. In relation to the inorganic poisons—the mineral especially—this change is much less likely to occur: probably it never occurs except as the result of post-mortem putrefaction.

5. *Decomposition, and loss of the poison in the dead body.*—It cannot be doubted that during the putrefactive changes which occur after death, some poisons may become altered, and even entirely disappear. Such a change would be most naturally expected in the organic poisons. With regard to the mineral poisons, although they may undergo chemical transformation after death, the *metal* is indestructible: accordingly, it may be found in some new combination. Thus, arsenious acid after a time is transformed into the yellow sulphide, through the agency of sulphuretted hydrogen, the product of putrefaction. The salts of iron are, by the same agent, along with ammonia, converted into the black sulphide. Corrosive sublimate, by the same means also, changes into the black sulphide. Among the organic poisons, cantharides and strychnia appear to possess remarkable powers of resistance to putrefaction.

The above reasons are amply sufficient to account for the failure of chemical analysis to detect many poisons in the body after death. But in every case of alleged poisoning—fatal or otherwise—where there has been a failure of chemical proof, the prosecution must be prepared to account *satisfactorily* for this failure; or else it will very materially aid in the acquittal of the accused.

Objects of the chemical analysis.—These are: 1. To ascertain the *nature* of the poison, and the probable *quantity* administered. If the analysis should discover a different poison from the one mentioned in the indictment, this is not thereby vitiated. The *quantity* administered can hardly ever be determined more than approximately; but as close an estimate as possible should be made by a careful weighing, or measuring, of the solid or liquid material submitted for analysis. The malicious intention of a prisoner may often be inferred from the quantity of poison discovered in the substance administered. For instance, if but a few grains of oxalic acid were detected in a large quantity of some drink, a conviction for an attempt to poison would hardly follow, since so small a quantity would be harmless; but if a large quantity of this acid were discovered, the motive of the act would be apparent.

There is a very common fallacy connected with the quantity of poison found in the stomach after death, to which allusion has already been made (*ante*, p. 26): the question is constantly put to the witness on a trial, whether the quantity found in the stomach was sufficient to cause death? The proper answer to this question is that the poison found in the stomach—whether in large or small quantity—has nothing whatever to do with the fatal result: it is merely the *surplus* of that which has been the real cause of death. Hence, to argue that, because only a very small amount of a poison has been discovered in the stomach (which amount was insufficient to cause death *in another person*), therefore the deceased could not have been poisoned, would be a most serious error. As has been already explained, death may result from poison, and not a particle of it be discovered in the stomach afterwards, simply because it has either been all removed by vomiting, or else (unless the dose was excessive) it has all disappeared by absorption. This principle concerning the residual poison found in the stomach after death, cannot be too thoroughly understood.

In case the poison discovered, either in the stomach, or in the absorbed state, be in very small quantity, it may always be plausibly urged by the defense that it was either *accidentally* introduced, or else had been taken *medicinally.* On the other hand, the presence of a *very large* quantity might suggest the idea of suicide, rather than of homicide, since it might seem to indicate a deliberate and determined purpose on the part of the deceased, and to be inconsistent with the theory of homicide. But caution should be observed in relation to such an admission, especially in connection with arsenic, since a case of murder has been recorded by Sir R. Christison, in which nearly one hundred grains of arsenic were found in the stomach after death. In the celebrated case of Madeline Smith, tried for poisoning L'Angelier with arsenic (Ed. Court of Just., July, 1857), the defense made a very strong point in the fact that *eighty-eight grains* of arsenious acid were discovered in the stomach of the deceased—an amount up to that time unheard of in a case of homicide, and therefore one which justified rather the idea of suicide.

2. Another important object of the chemical analysis is *to discover the administrator.* This is often accomplished by a careful analysis of certain implements, articles of food, clothing, cups, etc. The discovery of poison in connection with these may lead to the identification of the true culprit. (See Taylor, On Poisons, p. 188, for some striking illustrations.)

Certain precautions to be observed in performing the analysis.—Before commencing this, it is desirable that the toxicologist should inform himself, as far as possible, of the nature of the symptoms and (in a fatal case) of the post-mortem appearances observed in the suspected case. Such a knowledge will generally serve at least to put him on the proper track, by indicating to what particular class of poisons he should direct his researches. If, for instance, the symptoms clearly indicated one of the irritant poisons, the analyst would save himself much labor by first directing his examination in that particular line, without making an indiscriminate search for poisons. For the same reason, if the symptoms had been clearly those of a neurotic poison, his investigations should first be directed to that especial class.

It is a good rule to observe in the analysis of complex organic materials, to reduce the quantity of the liquid, by evaporation, to the smallest bulk compatible with the application of the tests to be applied. No doubt, a failure to detect the poison is often dependent upon a disregard of this very precaution, since a very small quantity of a poison diffused through a large amount of material may fail to be recognized by the appropriate tests; whereas, if properly concentrated, it would have been detected. Again, the action of chemical reagents is more or less modified by the quantity employed. In some instances, a very slight excess of the reagent may entirely prevent the formation of a precipitate which would otherwise appear. On the other hand, a deficiency in the quantity of the reagent may materially modify the result: thus, a small quantity of sulphuretted hydrogen produces, with a solution of corrosive sublimate, a *white* precipitate; while a large amount of this gas will occasion a *black*-colored deposit; and an intermediate quantity will produce a *red* precipitate.

Accuracy of the analysis.—In order to secure accuracy of result in the chemical analysis, several things are necessary: 1. To employ only a portion—usually one-half—of the material to be operated on; the other portion is kept in reserve, in case of accident, or for subsequent experiments. 2. The experimenter should never be satisfied with the result obtained from a single test, or from a single line of testing. The suspected substance ought to respond to *every* known test, or the reason for its not doing so should be distinctly explained. Thus, in testing for prussic acid, the analyst is not justified in stating, as was done in the Schoeppe case, that this poison was present, simply because "faint traces" were observed by the application of only *two* out of the four recognized tests for this substance, and one of these (the iron-test) very doubtful, inasmuch as no actual *precipitate* of Prussian blue took place, but only "a bluish coloration." We think it is the duty of the toxicologist always to employ the various corroborative tests, no matter how superfluous this may appear in *his own* judgment; by omitting to do this, his otherwise excellent evidence may be materially weakened.

In all cases of poisoning by the metallic compounds (arsenic, antimony, mercury, copper, etc.), we deem it essential to obtain the *metal;* and that, too, in such quantity as to enable it to be subjected to all the corroborative tests. Fortunately for the ends of justice, this can generally be accomplished without much difficulty. The production of the *metal* in court, together with the recognized results obtained by its manipulation, is justly regarded as the very strongest chemical evidence in cases of alleged poisoning by arsenic, tartar emetic, corrosive sublimate, etc. There is more or less fallacy in most of the other chemical proofs usually brought forward in cases of metallic poisoning; some plausible objection may, at least, be urged against them, under different circumstances, which must weaken their force as evidence; whereas, the production of the *metal* in tangible quantity is a proof that cannot be set aside. It should, therefore, always be rigidly insisted on in criminal trials.

In relation to the necessity for *obtaining the metal* in cases of metallic poisoning, which we have so strongly insisted on, it may be proper to remark that Orfila is not quite in accord with M. Devergie, who asserts "that it is a principle in legal medicine *that admits of no exception*, that the *extraction of the metal is required* in all cases, in order to prove the presence of a metallic poison." Orfila, in reply, asks how it would be possible to obtain the metal in cases of poisoning by the salts of potash, soda, baryta, and lime—all of which have a metallic base. He then proposes the question, whether the submitting a suspected salt of copper to *all* the well-recognized tests, and finding it to respond properly to all these, would not be in effect as strong a proof of the presence of copper as the production of the metal itself. In answer to the above, we would say that in speaking of *metallic* poisons the phrase is understood to apply to the *metals proper*, and not to the metals of the alkalies, earths, or alkaline earths. Orfila himself subsequently modifies Devergie's assertion, so as to admit the necessity of isolating the metal, in cases where any doubt arises, especially from the presence of coloring-matters, which might obscure the precipitates. (Toxicologie, i. pp. 6, 7, 8.)

3. Too much reliance should not be placed on the mere *colors* of a precipitate; and this for several substantial reasons. In the first place, colors are not always seen alike by different persons: M. Devergie remarks (Méd. Légale, iii. p. 17) that "nothing is so deceitful as an absolute reliance upon color in testing. Four persons may look at the same colored product, and it will be found to present to each a different shade or tint." Many illustrations of this might be adduced. In 1817, Donnall was tried for poisoning his mother-in-law with arsenic. The chemical tests relied upon for proving the presence of this poison were the two liquid *color*-tests (the ammonio-sulphate of copper and nitrate of silver), which threw down from the boiled contents of the stomach the appropriate green and yellow-colored precipitates. This was considered conclusive evidence; and a conviction of the accused would certainly have followed, if it had not been shown by another medical man that a decoction of onions would yield with the above-named tests precisely similar colored precipitates! (Beck's Med. Jurisp., ii. p. 580.) At one time, the red color obtained by the action of meconic acid on a persalt of iron was considered as the most reliable test for the presence of opium; but it has since been ascertained that the saliva contains a principle (sulpho-cyanide of potassium) which gives to the iron-salt a similar color! Dr. Taylor alludes to a case where morphia was believed to have been eliminated in the urine, because the iodic acid test produced with the latter the characteristic reaction; but it was subsequently ascertained that uric acid and urate of ammonia (constituents of healthy urine) will give precisely the same reaction. (On Poisons, p. 193.) Even the well-known and characteristic color-test of strychnia, which is not imitated by any other known substance, should not be *exclusively* relied upon in a criminal case, but ought to be corroborated by other tests.

In employing the sulphuretted hydrogen test for the metallic poisons, it is the *color* of the precipitate which is generally considered so characteristic as a result. Now, when the experiment is made with *perfectly pure* solutions of the metallic salts, the color of the precipitated sulphide may be accepted as a good criterion; but in the presence of *organic matter*, with which the mineral poison must necessarily be associated when extracted from a human body, it should never be forgotten that this test cannot be relied on exclusively; for, in the first place, the presence of organic matter will more or less mask the true color of the precipitate; and secondly, there are certain kinds of organic matter, combined with coloring materials, which, without the presence of any metallic salt, in an acid, or even in a neutral solution, will yield with sulphuretted hydrogen colored precipitates bearing strong resemblance to those above mentioned. How dangerous, then, would it be, in a medico-legal case, to rely exclusively upon this test for the purpose of determining the presence of a metallic poison! As if the more clearly to show the impropriety of this partial and exclusive mode of dealing with the metallic poisons, it so happens that this colored precipitate procured from complex organic material, and *containing no metallic substance whatever*, will behave, with the usual reagents, in a manner strikingly similar to that which has been supposed to be peculiar to the metals. (*Vide infra*, ARSENIC and ANTIMONY.)

The toxicologist should always be prepared to give a *positive* opinion in relation to the results of his analysis. Either he has, or has not, discovered the poison alleged. The *certainty* of this result in no wise depends upon the *quantity* found; for this can often be as well established by a fraction of a grain, as by a pound, of the substance,—we mean, of course, the *mere presence* of the poison. The question, how the presence of a minute quantity of a poison might be satisfactorily accounted for, is quite another affair, with which the analyst has no concern. The witness should avoid the use of such expressions as "feeble traces" or "mere traces" of poison, since these indicate a doubt in his own mind of the presence of the poison. However minute the quantity discovered may be, there should not be the shadow of a doubt of *its actual presence*. If he has really any doubts about his results, he ought frankly to say so.

One or two other points require consideration here. The analyst should not forget that while the minutest portion of some poisons—as *e.g.* the one-hundred-thousandth of a grain of strychnia—may be detected *in the pure state*, the result is very different if it is mixed with complex organic matters: in the latter case, the difficulty of discovering it is vastly increased, and the minimum quantity capable of detection becomes very considerably augmented. In all cases where the quantity of material is limited, it is advisable to apply the most characteristic test first, and then follow with the other corroborative tests, as before mentioned. To draw any positive inferences from a single reaction would be manifestly very unsafe, since a similar reaction is often produced by very different substances. For example, a slip of bright copper, if boiled in a hydrochloric acid solution of either arsenic, mercury, antimony, or of several other metals, and indeed in certain complex *organic* solutions acidified, will receive a dark-colored stain. To infer the presence of any of these metals, or indeed of any metal, from this one single reaction, would be a most serious error: a second experiment is needed. On heating the dried coated copper in a reduction-tube, a sublimate of octahedral crystals will indicate arsenic; whilst mercury is the only substance that will yield a sublimate of metallic globules; and a mere amorphous deposit in the tube would indicate organic matter. A *third* experiment, performed upon these deposits, would bring out the proof with still greater precision.

The great leading idea that ought to govern the toxicologist in all his chemical investigations is, not to see with *how small* a number of tests he can prove the presence of the poison sought for, but rather, *how many different tests* he can adduce to prove his point. I have known a veteran chemist of nearly fifty years' experience in public teaching, rather boastfully to assert on the witness-stand that, in making an analysis for poison, in a capital case, if he had "satisfied himself" of its presence by one or two tests, any further experiments were quite unnecessary. It did not seem to occur to him that further experiments might be required for the purpose of "satisfying" other people!

Poisonous impurities in tests and apparatus.—One of the most important cautions to be observed by the toxicologist in his researches, is to obtain *perfectly pure* reagents—a matter often of considerable difficulty. In conducting an analysis in a poison-case, where the question of life or death may be involved, the most scrupulous care must be used to guard against all possibility of error in this direction. It is not sufficient that his reagents have been procured from standard sources and are marked "chemically pure:" before using them, the analyst should each time test them for himself; otherwise he cannot *swear positively* to the purity of his reagents. Another caution just here may not be amiss: in these delicate manipulations, the analyst should avoid using the same reagents, bottles, etc., which he is accustomed to employ in the ordinary experiments of his laboratory and class-room. It is very evident that in the latter employment they must be constantly exposed to the action of foreign impurities, in the form of vapors, etc., and also be liable to constant indiscriminate handling. The *rule* to be sedulously observed is to guard against every *possibility* of impurity.

Very many of our ordinary reagents contain impurities: sulphuric acid is often contaminated with *arsenic* and *lead;* hydrochloric acid, with *arsenic* and *antimony;* nitric acid, with *iron* and *sulphuric* acid; zinc, with *arsenic.* Solutions of potash and soda are apt to contain traces of *lead* if kept in flint-glass bottles; *lead* is also found in carbonate of ammonia, citric acid, tartaric acid, bicarbonate of soda, etc. A moment's consideration will show the importance of attention to the above facts, since carelessness or ignorance on the part of the analyst might thus lead to the most serious results.

SECTION V.

EVIDENCE DERIVED FROM EXPERIMENTS ON ANIMALS.—PHYSIOLOGICAL PROOF.

IV. In certain cases, where the symptoms, the post-mortem lesions, and the chemical analysis fail to establish the positive proof of poisoning, we may with great advantage resort to the *physiological* evidence, or that derived from direct experiments on living animals. The animals best adapted to this purpose are the dog, the cat, and the rabbit—especially the former; and, in certain cases, the frog. *Birds* are particularly unsuited for experiments of this sort, since they are affected so very differently from man and the animals just mentioned. The exact sort of information obtained by such experiments seems to be limited to proving *the fact* of poisoning: we can gather no certain data from them relative to the dose, the rapidity of absorption, the deposition or elimination, in reference to man. We may also, occasionally, learn something of the physiological and pathological action of poisons. The instances of the remarkable discrepancy in the *dose* of poisonous substances necessary to produce death in man and the lower animals, are numerous. On one occasion, I administered to a dog, subcutaneously, fourteen grains of atropia, in divided doses, without fatal effect—a result that entirely confirms the experiments of Dr. Frazer on dogs and rabbits. Moreover, it should not be forgotten that there are some poisons, derived chiefly from the vegetable kingdom, that prove quite innocuous to certain animals, although very dangerous to man: among these may be mentioned the *datura stramonium*, which is eaten with impunity by cows and goats; the rabbit likewise will thrive upon the leaves of belladonna, stramonium, and hyoscyamus.

Important evidence of this kind is sometimes accidentally obtained, which may prove valuable as confirmatory proof of the administration of poison. Dr. Taylor (On Poisons, p. 196) mentions some instructive cases. A woman poisoned her husband with arsenic mixed with soup, and threw the remains of the meal out of the window into a farm-yard, thinking thus to get entirely rid of it. It happened at the time that a pig and several fowls were feeding under the window, and they ate up the food as it fell to the ground. The whole of these animals died under symptoms of irritant poisoning. The husband also died; no poison was detected in his stomach, although there were traces of its action; but on opening the bodies of the animals, not only were the usual appearances produced by irritant poisons found, but arsenic itself was also readily detected in the viscera. The prisoner was convicted and executed. In another case, it was proved that the prosecutor, to whom, it was alleged, the prisoner had given arsenic, went out into a back yard and vomited the food. Some fowls near the spot were observed to be ill during the day, and two died. The prisoner had, in the mean time, thrown away the poisoned food, and washed out the vessels which had contained it. As the prosecutor recovered, there could be no examination of his body. Arsenic, however, was found in the crops of the chickens which had fed at the spot where the prosecutor had vomited, and this supplied sufficient proof of the cause of his illness. Sometimes good negative, as well as affirmative, evidence, may be obtained by the examination of the bodies of animals alleged to have been poisoned. A case in point is related, where a woman poisoned her uncle with arsenic. When required to account for the poison that was found in her possession, she pretended that she had bought it for the destruction of vermin, and actually pointed out a dead mouse, in corroboration of her statement. This turned out to be an unfortunate part of her defense, for the medical witness showed that the mouse had not died from the effects of arsenic (*loc. cit.*).

Orfila, who devoted considerable attention to this subject, and made numerous experiments upon dogs with a variety of poisons, gives the following as the *résumé* of his deductions: 1. "Physiological experiments are unnecessary where the presence of the poison can be demonstrated by the chemical analysis. 2. When the chemical researches are unsatisfactory, and there remains some of the suspected material that has not been operated upon, this may be introduced into the stomach of a dog, and its effects noticed. 3. This experiment ought never to be performed with the material that has already been submitted to the action of chemical reagents, inasmuch as these latter might vitiate the results. 4. If the suspected matter occasion the animal's death, before concluding that there had been poisoning, we must be sure that the person, in whose alimentary canal it was found, had not died from some disease in consequence of which the animal fluids, and particularly the bile, have become infected and capable of producing many of the symptoms of poisoning. 5. In case the animal should exhibit no very decided symptoms as the result of swallowing the suspected matter, we have no right to conclude from this single experiment that no poisoning had existed, since there may be many reasons why the matters found in the stomach and bowels of a person really poisoned should not prove poisonous to the animal: thus, the poison may have been decomposed in the stomach by the food, drinks, or by the animal tissues, and even may be combined with them; for example, sixty centigrammes of corrosive sublimate were swallowed by a healthy man; symptoms of poisoning were brought on, and death followed; the body was opened from thirty-six to forty-eight hours afterwards, and the matters found in the digestive canal were given to a dog, which, however, experienced no bad effects from them. This has very frequently occurred.

"It is evident that, in the case just mentioned, the corrosive sublimate had been converted by the alimentary matters, and even by the mucous membrane of the stomach, into an insoluble substance, which exerted no noxious influence on the animal economy. A similar result may happen if verdigris (subacetate of copper) is taken either before or after swallowing albumen, and some other animal matters. Another reason may be assigned: the poison, although in large quantity, may have been expelled by vomiting, and death, nevertheless, have resulted: in such a case, the contents of the alimentary canal, being freed from the poisonous matter, would fail to occasion the death of the animal that might swallow them. Again, it might happen that the poisonous substance may be one that is easily absorbed; that although the deceased may have taken a large dose, only a very small portion remained in the stomach: here, the negative results obtained by the experiment on a dog, would lead to an erroneous conclusion." (Toxicologie, 1852, i. pp. 52, 53.)

Orfila recommends tying the œsophagus, in these experiments; assigning as a reason the great difficulty of making the animal swallow the suspected matters, in consequence of his resistance, and his quickly rejecting them. He states, moreover, that in the attempt it will happen at least once in ten times, that a portion of the material will flow back into the larynx, and the animal will perish from asphyxia (*loc. cit.*).

The best method to pursue, if the suspected material is in the liquid form, is to detach the œsophagus of the dog, while fasting, from the surrounding parts, and to inject the liquid into the stomach by means of a gum-elastic tube; then to tie the tube, and so to leave it for twenty-four to thirty-six hours. If the material is too thick to be introduced by means of the tube, the œsophagus, after being detached, should be pierced with a small opening, a glass funnel placed in the opening, and the matter introduced through it. The œsophagus should then be tied below the opening. If the suspected matter is in the solid form, so that it cannot pass through a funnel, it should be inclosed in a little roll of tissue-paper, and then pushed into the stomach, through the opening made in the œsophagus; and then this tube should be ligated as before.

Of course, the main object of tying the œsophagus, in these experiments, is to prevent the animal from vomiting, and to insure the retention of the substance injected sufficiently long for it to produce its usual effects upon the system. Many objections were made to this method of experimenting, even in Orfila's time; and these still continue to be urged, though, we think, without sufficient grounds. It has been stated, for instance, that the operation of tying the œsophagus is a hazardous one, and that the effects ascribed by Orfila to the alleged poison were, in reality, due to the operation. This objection is completely refuted by Orfila himself. He experimented over fifty times on dogs, often in the amphitheatre of the faculty, in the presence of members of the Academy of Medicine, and with the most positive results. As he rightly observes, the operation must be performed by a skillful hand; in which case, it need never occupy over two minutes. The œsophagus should be carefully separated from all its attachments—trachea, vessels, and nerves—and then tied. When the ligature is left on for twenty-four to thirty-six hours, the animals merely experience a slight depression, and a little fever; as soon as it is removed, they at once eat and drink, and seem perfectly at ease. The wound heals in a few days, without any further attention.

From the experiments of Orfila, I think the following conclusions warrantable: (1) Tying the œsophagus, even after first opening it, occasions during the first two days merely a slight fever and depression, incapable of causing death in that length of time. (2) If the animal be killed during this time, no pathological lesion is discoverable. It is hence evident that if an animal, caused to take poison a short time before tying his œsophagus, should die in the course of two days, after exhibiting severe symptoms, such as vertigo, convulsions, pain, or insensibility, efforts at vomiting, etc., the death should be attributed solely to the poison administered. What is absolutely confirmatory of this proposition is the fact that if another animal, whose œsophagus is not tied, be made to swallow an equal dose of the same poison (and do not vomit it), he will exhibit precisely the same results. These comparative experiments have been actually made in the case of strychnia, camphor, the upas tieuté, false angustura bark, and other substances *that were not vomited.* The inference, then, seems irresistible that the pathological changes observed in animals which have been poisoned, whose œsophagus was tied, and which have died within forty-eight hours after the operation, should be attributed only to the poisonous substance; since the operation could not have occasioned any alteration, except merely on the part cut. Any one can judge of the effect of the ligature on the œsophagus of all the animals to which poison was administered, and which died in *two*, *four*, *eight*, *twelve*, or *twenty-four hours after;* inasmuch as they comprise at least seven-eighths of those operated on.

Whilst it may happen, in cases where the poison *acts but feebly*, and life is prolonged for several days, that it becomes difficult, at times, to discriminate between the effects of the poison and those of the operation, such a difficulty could never happen in a case where the œsophagus is tied, *without previously opening it.* It is for this reason, chiefly, that the latter operation is to be preferred to that of opening the œsophagus before tying it.

The value of the *frog-test* in poisoning by strychnia will be more fully discussed hereafter. (See STRYCHNIA, *post.*)

Can poison be introduced into the human system through the body of an animal, without the latter being affected by it?—This is an interesting physiological question; and it may, under certain circumstances, assume an important practical aspect, as where serious symptoms have followed the eating of game that had fed on poisonous vegetables. The rabbit, for example, according to M. Runge, of Berlin, will eat, and thrive upon, the leaves of belladonna, hyoscyamus, and stramonium; although, on killing the animal, the absorbed poisons may be discovered in its body. The goat and cow will eat the leaves and stalk of the stramonium with perfect impunity, yet their milk may prove poisonous to those who drink it. One insect at least is known that can feed and flourish on strychnia. The fact is undoubted that, while certain animals, birds, and insects can eat poisonous plants with perfect impunity, the flesh and secretions of these creatures prove highly poisonous to human beings. Thus, the honey of the bee fed on the kalmia, azalea, and rhododendron, and even the mead made from it, has been found to be poisonous (Guy's Foren. Med., p. 350). Herodotus states that during the celebrated retreat of the ten thousand under Xenophon the army suffered greatly from using honey collected from the *Azalea Pontica.* The flesh of hares that had eaten the *Rhododendron chrysanthemum*, that of pheasants that had fed on the buds and shoots of the *Kalmia latifolia*, and that of partridges that had partaken of certain berries during the Canadian winter, and had been imported into England packed in ice, have proved poisonous. This very question was made a ground of defense in the case of Sprague, tried for attempting to poison the Chalker family, and acquitted. It was urged that the belladonna (the alleged poison) had found its way into the poisoned pie through the flesh of a rabbit that had fed on the plant (Guy's Forensic Med., p. 349). The "Medical Times and Gazette," Sept. 13, 1862, contains an account of several persons near Toulouse who were poisoned by a dish of snails that had fattened on the leaves and shoots of the *Coriaria myrtifolia.*

SECTION VI.

EVIDENCE DERIVED FROM THE CIRCUMSTANCES.—MORAL EVIDENCE.

V. The circumstances attending death by poisoning, like those connected with any other violent death, may occasionally throw much light upon the case, so much so indeed as to afford most of the required proof of guilt. Although in criminal trials, as a general rule, the expert witness has nothing to do with this sort of evidence, nevertheless, in poison-cases particularly, the medical and moral circumstances are often so interwoven that, in order rightly to appreciate the latter, and assign to them their proper weight, the aid of the medical witness is invoked. Hence the importance of his proper understanding of them.

1. The first of these "circumstances" to be noticed is *the suspicious conduct of the accused before the event*, such as dabbling with certain poisons not in the way of his profession, conversing about them, experimenting with them, etc. This was a very strong circumstance against the Count Bocarmé, who was convicted of poisoning his brother-in-law, M. Fougnies, in Belgium, by forcibly introducing *nicotina* (the active principle of tobacco) into his throat. It was proved that the prisoner had given his special attention and study to the most approved methods of distilling this (then) almost unknown poison; that he had a considerable quantity of it in his possession; that he had carefully concealed his chemical apparatus; that he used an assumed name in his different purchases, etc.

2. The *purchase*, or the *possession*, of the alleged poison just before the event; the procuring of it secretly, or under false pretenses. This is a very common and familiar circumstance in trials for poisoning, and it generally carries much weight against the prisoner. In the alleged purchase of arsenic, or strychnia, for the destruction of rats, it would be a very suspicious circumstance if the purchaser had not apprised his household of the fact, and warned them against taking it by mistake.

3. *The proof of actual administration* of the poison in food,

drink, or medicine. This proof is very seldom attainable by direct evidence; but it may be often brought home to the guilty party by a chain of circumstances of a strictly medical nature. William Muir was condemned at Glasgow in 1812 for poisoning his wife. She sickened soon after breakfasting on some porridge, which she suspected had been poisoned. Soon after, a neighbor who had some knowledge of medicine called in, and, hearing the circumstances, examined the wooden bowl containing the remnants of the oat-meal from which the breakfast had been prepared; this was found to contain shining particles, which subsequently proved to be arsenic. He also examined the barrel containing the family store of meal, which proved to be free from the poison. This circumstance was enough to show that the poison had been mixed with the meal, intended for the woman's breakfast, and on that very morning, before any stranger had entered the cottage, and consequently to fasten suspicion very strongly upon the husband, the only other person in the house.

A similar case is related by Dr. Taylor (Hartley's case), where a girl was accused of attempting to poison her father by putting oil of vitriol into his coffee. Here the point to be established was, could anybody else besides the prisoner have put the poison into the coffee? She had the opportunity to do it, as also to put it into the coffee-pot at the time she prepared it; whilst others might have put it into his cup after it was poured out. Dr. Taylor ascertained that the coffee-pot was old and rusty; the poisoned coffee in the cup contained no trace of iron (which it would have done if the acid had been put into the coffee-pot); whereas when a portion of the poisoned coffee was warmed in the pot, it immediately became impregnated with sulphate of iron. This analysis satisfactorily established the fact that the poison had not been put into the coffee-pot, but into the cup; and to this, others besides the prisoner had access. In Humphrey's case, tried at Aberdeen, in 1830, the charge of poisoning her husband by pouring sulphuric acid down his throat while asleep, was rendered all but positive by the detection of stains of this acid on the prisoner's bed-gown and handkerchief. In the case of Reg. *v.* North (Guilford Summer Assizes, 1846), the prisoner was tried for administering oil of vitriol to a young infant. The circumstance that most strongly implicated her was of a negative character. She was proved to have carried the child into a closet where she kept a bottle of oil of vitriol; and when she returned, the infant was writhing in great pain, and the mouth covered with a white foam. The accused alleged that the mother of the child had administered the poison by mistake for a composing draught; but, if this had really been the case, there would have been a blacking of the white sugar in the cup, when the oil of vitriol was poured upon it, *which did not occur:* consequently, it was impossible that the mother had made the alleged mistake.

4. *The intention of the accused;*—the possibility of his having administered the poison ignorantly or unintentionally.—This plea is sometimes urged by the defense; but generally the particulars of the case will serve to expose the falsehood. Mr. Hodgson, surgeon, was tried for attempting to poison his wife, in 1824, by substituting corrosive sublimate in place of some pills of calomel and opium that had been prescribed for her. In his defense, he pleaded intoxication, which had caused him to mistake the bottle when he prepared the pills. On another occasion, when the physician sent him to compound a draught of laudanum and water, he brought back a turbid mixture, which the physician found to contain corrosive sublimate. The prisoner again attempted to defend himself by alleging a second mistake,—that he had inadvertently substituted for water a solution of corrosive sublimate of a certain strength, which he kept in his shop for a special purpose. The falsehood was shown by the fact that while the latter solution contained but five grains of the salt to the ounce of water, the former contained fourteen grains.

5. *The simultaneous illness of other persons* who had partaken of the same articles of food or drink, besides the one more particularly affected; the researches and opinion of the medical witness are required to substantiate this circumstance.

6. *Suspicious conduct of the accused during the illness and after*

the death of the deceased.—Many circumstances may occur in this relation which may contribute largely to fix suspicion on the guilty party, and which the medical witness may aid materially in bringing out: such as directly or indirectly preventing medical advice being obtained, or the relatives of the dying person being sent for; taking the exclusive care of the patient, and showing an over-anxiety not to leave him alone with another person; allowing no one else to give the food or medicine; carefully removing the remains of his food, drink, and medicine, and also the matters vomited, and the excreta; and expressing the opinion of a speedy death; after the decease, interposing objections to a post-mortem examination of the body; and, if this is insisted on, taking care to spoil the result as far as possible, by spilling the contents of the stomach, as if by accident (as was done in the Palmer case); hastening the funeral, and giving a false account of the illness.

7. The *existence* of a *motive*, or *inducement*, on the part of the accused, such as a desire for revenge, or the expectation of inheriting property by the death of the party (as in insurance-murders); the desire to be relieved of a debt that has been long pressing for payment by the deceased, etc. The latter was illustrated in the celebrated Webster trial in Boston, in 1850, where the prisoner was convicted of having killed Dr. Parkman (though not by poison), to whom he had been for some time indebted for a considerable sum of money.

The above comprise the most common and the most important points of circumstantial evidence that are likely to be brought out at a trial for poisoning. They are substantially the same as those contained in Christison "On Poisons," to which work the reader is referred for fuller details. It would be well for physicians who may be consulted as "experts" in such cases to bestow the proper attention to these various "circumstances," as they present themselves on different occasions.

CHAPTER VI.

COMPOUND POISONING.—ANTAGONISM OF POISONS.

The subject of Compound Poisoning is one of much interest, as likewise of considerable importance in its medico-legal bearing. In a case of alleged criminal poisoning, the question might arise whether the usual symptoms may not be so modified, or masked by the presence of some other poison, as to escape detection; and further, whether, after death, the usual morbid appearances occasioned by one or both may be modified, or perhaps be altogether absent. Our knowledge relating to this subject is comparatively limited, at least so far as the human system is concerned. Within the last few years, a number of experiments on the lower animals have been performed for the purpose of determining the question of the antagonism of poisons, the results of which seem to show that in some instances at least, and up to a certain limit as to dose, this antagonism really does exist in the case of some of the inferior animals. Dr. Frazer has demonstrated this conclusively in the case of *atropia* and *physostigmia* (the active principle of Calabar bean), on the rabbit (Trans. Roy. Soc. Edin., vol. xxvi.).

Sir R. Christison (On Poisons, p. 970) mentions several cases that have fallen under his own notice, where the usual effects of certain poisons appeared to be decidedly modified by the presence of some other poison which had been taken simultaneously, or just before, or subsequently. One instance is that of a man who, after taking twelve ounces of whisky at a debauch, swallowed, an hour afterwards, while partially drunk, a quantity of arsenic, the dose of which could not be ascertained. Fifteen minutes after the arsenic had been swallowed, medical aid was procured, when repeated but ineffectual attempts were made to vomit him. The stomach-pump was now resorted to, which removed a fluid from the stomach in which arsenic was easily discovered. No symptom of arsenic-poisoning followed, although this substance was taken on an empty stomach, seven hours after eating—when all the circumstances were favorable for absorption. Here, the inference would seem justifiable that the alcoholic narcotism did, in some way, suspend, or arrest, the usual operation of arsenic.

In another similar instance, death ensued in consequence of the large dose of arsenic taken. A lad aged seventeen years, after a night's debauch, swallowed half an ounce of arsenic in the morning. In two hours and a half afterwards, when seen by a physician, there was no symptom of arsenic-poisoning, but merely drowsiness and languor. Shortly afterwards there was slight vomiting, which, however, required to be artificially renewed. In eighteen hours he began to sink, and then presented the usual constitutional symptoms of poisoning with arsenic, and in forty-one hours he expired. From first to last, he showed scarcely a single local symptom, except the slight vomiting, although after death the stomach presented signs of violent irritation.

From the above, and similar cases that have come to our knowledge, we think there can be no doubt that alcoholic intoxication has the effect of obtunding the system to the action of irritant poisons; and to such an extent as often to neutralize their operation, unless the dose has been extremely large, and there has been little or no emesis to remove the poison from the stomach.

Another remarkable instance related by Christison (*loc. cit.*, p. 972) was that of a young soldier, who swallowed a mixture of *corrosive sublimate and laudanum*—two drachms of the former and half an ounce of the latter. He at first had no violent symptoms whatever to indicate the ingestion of corrosive sublimate,—a very uncommon occurrence. Afterwards there was purging of bloody stools, with tenesmus, but no abdominal pain; no tenderness even on pressure; and no vomiting except under the use of emetics. On the fourth day, salivation set in, and under this and the dysentery he finally sank, dying on the *ninth* day after the attack, quite unexpectedly. The stomach and bowels were found enormously inflamed, ulcerated, and almost gangrenous. It can scarcely be supposed, as Christison remarks, that the laudanum acted here as a chemical antidote, and that an insoluble *meconate of mercury* was formed. We must rather ascribe the results to the protective power of the narcotic.

In order that this theory of the antagonism of poisons should have any weight, or be urged with any plausibility in a criminal trial, we think that the circumstantial proofs of the administration of the alleged combination should be most positive and unequivocal,—such as the purchase, possession, and actual administration, and especially, in a fatal case, the actual discovery, of one at least of the alleged poisons, after death. It would evidently be a most dangerous precedent to establish that, in a case of alleged poisoning, conviction should follow, in the absence both of the usual symptoms and of the usual post-mortem signs, as also where there is a failure of the ordinary chemical analysis; and with nothing to support the charge, except the plea of "compound poisoning"! Nothing could possibly warrant such a conclusion, except the most absolute proof of the administration of the "compound poison," and also an equally conclusive proof of the absence of all disease.

A most extraordinary instance of the attempt to urge this plea of "compound poisoning" occurred in the celebrated trials of Dr. Paul Schoeppe, at Carlisle, Pa., in 1868 and 1872, for the alleged poisoning of Miss Stennecke. The prisoner was first indicted for administering prussic acid to the deceased, his patient, who had died with all the symptoms of apoplexy, more than thirty hours after taking any food or medicine. There was not a single recognized symptom of prussic acid poisoning; nor indeed was the idea of poisoning entertained at all, until some days after her death, when a will was discovered, made by the deceased in favor of the prisoner, to the exclusion of her heirs-at-law. The body was then examined for prussic acid; but the method of analysis pursued by the chemist was so inconclusive (see Hydrocyanic Acid), that the Commonwealth abandoned this charge, and substituted another, viz., that of *compound poisoning* by prussic acid and morphia. Here comes in the most remarkable circumstance of this remarkable trial. As the plea of a "compound poison" was entirely novel in the criminal jurisprudence of our country, and no expert could be found to testify to its reality either from personal experience, or otherwise, the testimony of one of the medical witnesses for the prosecution was allowed to go before the jury, to the following effect: that, some thirty years previously, he (the physician) had experimented upon a chicken-hawk by giving it a mixture of prussic acid, corrosive sublimate, and laudanum, made up with bread-crumbs; that the bird died on the following day; and that after death, "its eyes looked just like the eyes of the deceased lady;" and *therefore* he concluded that her death had been caused by a mixture of prussic acid and morphia! For the credit of justice we regret to be compelled to add that this "expert" evidence was made to weigh so powerfully with the jury, that they actually convicted the prisoner of murder, and sentence of death was passed upon him; and he was respited only a day or two before the time fixed for his execution, in consequence of the strong and almost unanimous protest of the medical and chemical professions, throughout the country. For over three years the accused was allowed to remain in prison, when at length another trial was obtained for him, the result of which was a complete overthrow of the Commonwealth's previous "expert" testimony, and a prompt acquittal by the other jury who tried him.

In the year 1870 the author performed a number of experiments upon dogs, with the view of testing this question of the antagonism of certain poisons. A few of the results only will be here briefly mentioned.

1. *Prussic acid and morphia.*—The antagonism was found to be very slight, if indeed it existed at all. It was observed that when the dose of prussic acid was less than the minimum fatal quantity, and the morphia was in excess, the effects resulting were decidedly those of the latter agent. If, however, both poisons were given in full doses (two fluidrachms of prussic acid, and two grains of morphia), in divided portions, the symptoms of both toxic agents were

manifested, and with a fatal result. Evidently, the morphia did not counteract the fatal effects of the prussic acid; *and it never does, if the latter be taken in a full poisonous dose.* In another experiment, in which the quantity of both the poisons was just within the minimum fatal doses, the animal finally recovered, after exhibiting a true combination of symptoms,—sometimes those of the one poison following its administration, and sometimes those of the other.

2. *Morphia and atropia.*—The first point to be noticed here is the remarkable tolerance of the dog for atropia: doses of *eight* and even *fourteen* grains did not destroy life; and doubtless a larger dose might have been borne. The tolerance of the rabbit is even greater. Dr. Frazer in his experiments found that the minimum fatal dose of sulphate of atropia for a rabbit weighing three pounds, was *twenty-one grains.* It is well known that this animal will actually fatten on the leaves of the belladonna. Birds also appear to enjoy the same immunity from its poisonous impression.

The most observable effects on dogs were partial or complete loss of muscular power (paralysis), with only slight nervo-excitant effects, such as twitchings, but no distinct convulsions; dilatation of the pupil, and blindness; with dryness of the tongue and fauces. When the two poisons were given in combination, the results of the experiments did not indicate any real antagonism. In one of these trials, in which four grains of morphia were injected subcutaneously, narcotism was fully produced in one hour, when four grains of atropia were similarly administered; the animal became paralyzed in an hour, but showed no disposition to spasms. In twenty-four hours he had completely recovered, with the exception of a slight dilatation of the pupils.

In reltaion to the alleged mutual *antidotal* power of morphia and atropia in *man*, which seems now to be generally admitted, it must be remembered that we are not yet fully authorized to draw inferences from experiments on animals, in relation to the human subject; for the reason that the tolerance of the former for one, at least, of these substances —atropia—is so vastly greater than in the case of man.

3. *Strychnia and prussic acid.*—These two powerful poisons

exhibit no mutual antagonizing properties. When exhibited together in about the ordinary minimum lethal dose—one fluidrachm of prussic acid and three-quarters of a grain of strychnia—the immediate effect was that of the former poison, such as panting, respiration, great dilatation of pupils, inability to stand, and convulsions. This was followed in four minutes by the tetanic spasm of strychnia; and a succession of ordinary convulsions and tetanic spasms, alternating, ensued, until the animal died, eighteen minutes after receiving the poisons. In other experiments in which the substances were administered together, in rather smaller quantity than the minimum fatal dose of each, death ensued,—the symptoms of each being perfectly distinguishable, alternating with one another. Certainly there was no real antagonism between them.

4. *Strychnia and morphia.*—These alkaloids showed no evidence of any antagonistic power, at least in dogs. One of these animals was completely narcotized by the hypodermic injection of a grain of morphia; he was very drowsy; could not stand; pupils moderately dilated. Half a grain of strychnia was then given hypodermically, which manifested its effects in five minutes, and in another minute produced a decided tetanic convulsion, followed by others; death occurred in twenty-one minutes. Other experiments on cats indicated satisfactorily that morphia, so far from diminishing the power of strychnia, rather intensified it in this animal.

5. *Atropia and physostigmia.*—The interesting experiments of Dr. Frazer with these substances on dogs and rabbits (Trans. Roy. Soc. Edin., vol. xxvi.), demonstrate very clearly their physiological antagonism. A dose of physostigmia three and a half times greater than the minimum fatal, is completely neutralized by an exceedingly small (comparatively) dose of atropia—one-tenth to one-fifth of a grain. In treating a case of poisoning by Calabar bean in man, the atropia should be administered hypodermically—one-fiftieth to one-thirtieth of a grain; and this should be continued until its characteristic impression is produced, as indicated by dilatation of the pupils, etc. The action of these two substances

physiologically, is remarkably distinct, more so than that of any other known poisons.

6. *Atropia and strychnia.*—There would appear to be an antagonism between these two powerful poisons, judging from an interesting case reported by Mr. S. Buckley in the Edin. Med. Jour., Sept., 1873. A woman, aged twenty-eight years, had taken an unknown quantity of strychnia with a view to suicide. When seen half an hour afterwards, she was in a state of complete opisthotonos, the spasms severe and painful, and the intervals short. The stomach was washed out by the stomach-pump, and chloroform administered with a view to relieve the spasms, but without apparent effect. As an antidote, twenty minims of liquor atropiæ, equal to one-sixth of a grain, were injected subcutaneously three times, at intervals of ten minutes. "Under this treatment, a semi-comatose condition supervened, and after each injection the spasms became milder in character. At this period the heart's action was impetuous and irregular; the pulse 130, and fluttering; respiration hurried and somewhat stertorous; the pupils widely dilated; the face flushed, and the features fixed. The atropia was now continued, but in smaller doses, and at longer intervals. The spasms always increased when the injections were long omitted." Chloroform had been discontinued. Consciousness returned in about eight hours after the poison was swallowed; and, after sleeping a few hours, she appeared to suffer no further inconvenience except a sense of uneasiness about the throat, and, on the day following, some stiffness of the joints. The whole amount of atropia administered was one and a sixth grains—an enormous dose, when it is remembered that one-sixth of a grain has proved fatal under ordinary circumstances. (See articles by the author, On the Antagonism of Poisons, in Amer. Jour. Med. Sciences, Jan. and April, 1871.)

CHAPTER VII.

METHOD OF CHEMICAL PROCEDURE IN A CASE OF SUSPECTED POISONING.

THE following rules are taken mainly from the excellent treatise of Tardieu and Roussin (Sur l'Empoisonnement, chap. iv.). The two accompanying tables have been also translated from the same authority.

After being thoroughly satisfied in relation to the *identity* of the substances submitted for his examination (*ante*, p. 65), and having obtained all necessary information in relation to the previous symptoms, and (in a fatal case) the post-mortem appearances, as a guide for his manipulations, the toxicologist is prepared to perform the chemical analysis. This may be required not only for the viscera and organs of the dead body, but also for various substances discovered and seized by the officers of the law—such as suspected food and drinks, medicinal preparations, vomited matters, dejections from the bowels, urine, and any other matters supposed to aid in the research. In the great majority of cases the poisons to be sought for are those that are well known, and such as can usually be detected with certainty by the skillful chemist. Should, however, the poison be one of great rarity, and one whose analysis has not hitherto been attempted, the chemist must expect to encounter considerable embarrassment in his investigation, unless, by good fortune, some clew to its nature has been furnished him in advance.

In an examination of a mineral substance, even without any previous knowledge of its nature, a few preliminary experiments will usually lead to the discovery of its true character. But if the substance be of an organic nature, and especially if it be associated with complex animal matters, the analysis becomes far more complicated, and the reactions much less certain and definite. In the language of Tardieu: "Changeable and liable to decomposition to an unlimited extent, organic substances are endowed with great mobility, and are with great difficulty operated upon so as to obtain satisfactory results. Their reactions interfere with, confuse, and destroy each other; or they mask one another so as often to deceive the most skillful chemist" (*loc. cit.*, p. 62). Instead of pursuing a different *special* method for each distinct poison suspected to be present, the better course for the toxicologist is to adopt one or two of the well-established methods of procedure, which have received the sanction of long experience.

Before submitting the organs, etc., to chemical analysis, they should be subjected to a thorough *physical* examination, as already intimated (*ante*, p. 65). This may lead to the discovery of various matters in the stomach and intestines that may throw considerable light on the case—such as fragments of leaves and roots, seeds, woody fibre, granules of fecula, portions of powder or fragments of mineral substances, such as arsenic, corrosive sublimate, etc. The state of preservation of the organs should be carefully regarded, as giving, in certain cases, valuable indication of the nature of the poison. The *odor* exhaled from the interior of the organs will often indicate the nature of certain poisons—*e.g.* phosphorus, chloroform, nicotina, and prussic acid. The acid or alkaline condition of the material submitted should also be first ascertained, by means of litmus and turmeric paper.

As already mentioned, the main and primary object, in a search for poison in animal tissues and products, is to get rid of the organic matters,—an object which is sometimes very difficult to accomplish. Although several methods have been devised, at different times, for this purpose, two only will be here described, as being the most perfect and reliable. The first is that recommended by Fresenius and Babo. The solid matters are cut up into small pieces, which, along with the accompanying liquid, are to be put into a clean porcelain dish, and treated with a quantity of pure hydrochloric acid, somewhat greater in weight than that of the solid matter present, together with sufficient distilled water to form a thin paste. The dish, with its contents, is next heated on a water-bath, and about twenty grains of powdered chlorate of potassa added to the hot liquid, and this repeated from time to time with frequent stirring, until the solid matter disappears, and the mass becomes perfectly homogeneous, and of a light-yellow color. It is then to be heated until the odor of chlorine has entirely disappeared, a little water being added occasionally, to prevent concentration. When entirely cool, the liquid is strained through linen, any solid residue on the strainer being washed with warm water, and the washings, after concentration, added to the solution; the mixed liquids are next filtered through paper. Although a very large portion of the organic matter is destroyed and eliminated by this process, it is never wholly gotten rid of. Still, the ultimate concentrated and filtered solution can be employed in qualitative testing for the usual mineral poisons. It is not, however, sufficiently pure for accurate quantitative testing.

The second process is that devised by MM. Flandin and Danger, and is particularly recommended by MM. Tardieu and Roussin. It is the one generally adopted in France; and is named the process of *carbonization*, from the fact that the organic matters are reduced to charcoal, through the agency of pure sulphuric acid and heat. The organic matters, brought to the consistence of a soft extract by evaporation, are put into a tubulated retort attached to a double-mouthed receiver, along with one-fourth their weight of pure concentrated sulphuric acid, and the whole heated on a sand-bath for several hours, until the acid vapors cease to escape. After cooling, the carbonaceous mass is removed from the retort, and reduced to powder in a porcelain mortar. This powder is then treated with sufficient strong nitric acid, which will dissolve out all the mineral substances present, and afterwards with a little boiling water. The acid liquor is next to be evaporated to dryness, and the dry residue dissolved in distilled water. This solution will contain those metals the nitrates of which are soluble in water, together with the inorganic constituents of the animal materials operated upon, such as lime, magnesia, iron, alumina, soda, with phosphoric and hydrochloric acids.

If, for certain valid reasons, such as positive information relative to the nature of the poison, or the discovery of something in the preliminary examination, the analyst deem himself to be on the track of some special poison, he should appropriate a portion of the material—say one-fourth—for this especial investigation, which he may proceed to make at once; and if successful in the research, he may confirm his results by further experiments on the remainder of his material; or he may employ this (in certain cases) in the search for other poisons. But if the chemist has no clew whatever to indicate to him the particular line of investigation, it remains only for him to practice a careful methodical analysis, as follows:

He commences by dividing into two equal parts each of the suspected organs, and also the liquids in contact with them. These portions are put into two separate vessels, and are destined for two distinct analyses,—one for the search for mineral poisons, the other for that for organic poisons. These two separate portions are designated respectively by the numbers I. and II.

In the general method of research about to be described, the corrosive acids are not included, inasmuch as these produce such characteristic symptoms, and leave behind such well-defined marks of recognition, that it is useless to encumber our description with them. The proper method for their investigation will be detailed under their own special headings.

The two accompanying tables represent at a glance the methods of procedure in both cases. The details of the analysis will now be given.

TABLE 1.

The half (No. 1) of the organs, mixed with a fourth of its weight of pure sulphuric acid, is introduced into a Mitscherlich apparatus, terminated by a recipient, which contains a solution of nitrate of silver: this is subjected to a careful distillation(1).	The apparatus emits phosphorescent flashes in the dark (2)					**PHOSPHORUS.**
	The apparatus emits no flashes, but the distilled product yields with the nitrate of silver solution a precipitate insoluble in cold nitric acid, but soluble in boiling acid (3)					**PRUSSIC ACID.**
	The distillation in the Mitscherlich apparatus having failed to produce either flashes of light or precipitate, the original mass is transferred to a tubulated retort, properly fitted to a cooled receiver, and distillation by means of a sand-bath is practiced until dryness. The black carbonaceous mass is removed from the retort, and ground to powder in a porcelain mortar; it is then put into a vessel with a tenth of its weight of pure nitric acid, boiled for half an hour, diluted with warm distilled water, and filtered. To the clear filtrate ammonia is added till a white precipitate begins to form. A current of pure sulphuretted hydrogen is next passed through it to saturation; the liquid is allowed to stand twenty-four hours, at the end of which time there is, or is not, formed a metallic precipitate (4).	A metallic precipitate. . .	Black. . . .	It is collected and washed, and boiled for half an hour with pure nitric acid, in a porcelain capsule. The greater part of the acid is thus dissipated; the residue is dissolved in a small quantity of distilled water (5).	Dropped on copper foil, it causes a white stain, dissipated by heat (6)	**MERCURY.**
					Deposited on a bright strip of iron, it gives a red stain, colored blue by ammonia (7).	**COPPER.**
					Treated with a solution of iodide of potassium, it yields a yellow, or with sulphate of soda a white, precipitate (8)	**LEAD.**
			Yellow. . .	Soluble in ammonia. The yellow precipitate dissolved in boiling nitric acid, and put into a Marsh's apparatus, yields characteristic shining spots, which immediately dissolve in a solution of hypochlorite of soda (9)		**ARSENIC.**
		No metallic precipitate.	If sulphuretted hydrogen cause no precipitate, the liquid is evaporated to one-tenth of its volume, and introduced into a Marsh's apparatus (10) . . .	Metallic spots are obtained, soluble in hypochlorite of soda (11)		**ARSENIC.**
				No metallic deposit. In this case recourse is again had to the carbonaceous mass on the filter: this is divided into two parts:	The first is boiled for half an hour with pure carbonate of soda, filtered, and the charcoal washed with dilute nitric acid. The acid solution evaporated to a proper quantity gives with sulphuretted hydrogen a black, and with iodide of potassium a yellow, precipitate (12)	**LEAD.**
					The second part is boiled with tartaric acid, and the solution, put into a Marsh's apparatus, yields metallic spots, not soluble in hypochlorite of soda (13) . . .	**ANTIMONY.**

TABLE 2.

The half (No. 2) of the organs, etc., very finely divided and brought to the condition of a thin soup, is put into a tubulated retort, the end of which is fitted to a porcelain tube, which can be heated to redness, and which terminates in the Liebig's bulbs, containing a solution of nitrate of silver. To the tubulure of the retort is adapted a bellows, by means of a gum-elastic tube, through which air can be forced (1) . .	The nitrate of silver solution becomes clouded.	The precipitate is white, soluble in ammonia, and insoluble in boiling nitric acid (2)		Chloroform.
	The nitrate of silver solution is not clouded. . The contents of the retort are then to be treated by the process of Stas (3). The final residue of this treatment is:	Liquid.—Very alkaline; volatile—exhaling the strong odor of tobacco (4) . . .		Nicotina.
		Solid.—One portion of this is to be introduced into an incision made in a live frog; the following effects are produced (5):	Extreme dilatation of pupils.—The residue in this case dissolves easily in water, giving an alkaline solution, which yields a brown precipitate with iodine, easily changing, and giving out a nauseous odor (6)	Atropia.
			Violent tetanic convulsions occur, intermitting.—The residue in this case, moistened with strong sulphuric acid, on the addition of a fragment of bichromate of potassa, yields a violet color, which soon changes (7)	Strychnia.
			Rapid depression; intermittent and irregular action of heart.—The residue should be soluble in warm water, and the solution, although devoid of alkalinity, is precipitated by tannic acid. The residue is colored green by hydrochloric acid (8)	Digitaline.
			Complex physiological phenomena.—The residue in this case is crystalline; almost insoluble in water and ether; soluble in caustic potassa. It assumes a blue tint when it is thrown, in powder, into a concentrated and slightly acid solution of persulphate of iron; it instantly decomposes iodic acid; it gives an orange color to strong nitric acid (9)	Opium.

I. *Search for mineral poisons. Portion No. I.* (*Table* 1.)—(1) This half of the material should first be divided into small pieces by means of a knife or scissors, which should be thoroughly clean and bright. The pulpy mass obtained as above is first weighed, and then introduced into the apparatus of Mitscherlich (see *post*, PHOSPHORUS), into which pure and concentrated sulphuric acid equal to one-fourth the weight of the material is poured. If the matters contained in the retort are too thick, distilled water is added in sufficient quantity to insure distillation. The retort is put upon a sand-bath, and cautiously heated for half an hour.

(2) The appearance of phosphorescence in the course of the distillation will indicate the presence of *phosphorus*. In order to observe this, it is indispensable that the experiment be performed in the dark. (For details, see *post*, PHOSPHORUS.)

(3) The apparatus of Mitscherlich is made to terminate in a bent tube which enters a flask containing a solution of nitrate of silver, without, however, touching the solution. If a white precipitate occurs, it is probable that *prussic acid* is present. (For details and exceptions, see *post*, PRUSSIC ACID.)

(4) If the distillation causes neither phosphorescence, nor a white cloudiness in the silver solution, the apparatus is allowed to cool; and the contents of the flask are transferred to a tubulated retort well stoppered: these should be introduced through the tubulure, by means of a wide-mouthed funnel with a long neck. The flask and funnel should be washed off with a little distilled water, which is then added to the mass; this should not occupy over a fourth of the capacity of the retort, which should be fitted, by means of an adopter, to a large receiver, kept properly refrigerated. The retort is now placed upon a sand-bath, and its contents carefully distilled, the process being continued until these are dry. This operation is always tedious, and should be conducted slowly; the time ordinarily required is about six hours. When completed, and the apparatus is cooled, the distilled liquid is set aside; and, with the aid of a glass rod, the black and carbonaceous contents of the retort are extracted. (See *post*.) This substance is to be powdered gradually

in a porcelain mortar, and then introduced into a glass flask, along with a tenth of its weight of pure nitric acid, and allowed to digest for half an hour at the temperature of 100° C.; then a little boiling distilled water is added, and the whole is filtered. If the carbonization was complete, the filtrate will be colorless. (If it still retains a yellowish color, it must be evaporated to dryness, after having first added a little pure sulphuric acid; the residue must be again treated with nitric acid; diluted with water, and filtered the second time.) The charcoal is repeatedly washed on the filter by hot distilled water, and the washings added to the filtrate. This liquid is very acid: it contains a large quantity of sulphuric and nitric acids. After it is completely cooled, it is saturated with pure liquid ammonia, until a white precipitate begins to form. This liquid is next put into a flask which should be just large enough to hold it, and a current of washed sulphuretted hydrogen gas should be passed through it to saturation. The closed flask is left to itself for twenty-four hours; at the end of which time any metallic sulphides, which are insoluble in slightly acid liquids, will be completely precipitated.

(5) If the precipitate is black, or brownish-black, the supernatant liquid is carefully decanted by means of a siphon, and the flask is again filled with recently boiled distilled water. The decantation and washing are twice repeated, and then the precipitate with its adhering liquid is poured into a small porcelain capsule, and is dried upon a water-bath. To the dried residue several grammes of pure nitric acid are added, and the temperature kept to near the boiling-point, until all the acid vapors have disappeared; after which a small quantity of warm distilled water is added. (Liquid A.)

(6) A few drops of the preceding liquid are placed carefully upon a slip of bright copper. After a contact of twenty minutes, this is washed off with distilled water, and dried at a gentle heat. If the copper appears to be whitened, the spot is gently rubbed with the finger of a glove, to brighten it and make it more apparent. If the stain is due to *mercury*, it will readily disappear when put into the flame of a spirit-lamp. (See *post*, POISONING BY MERCURY.)

(7) Some drops of the same liquid (A) being put upon a strip of polished iron cause a red deposit, which is rather adherent, becoming brighter by washing and drying under a slight friction (*copper*). This red spot on being covered with a drop of liquor ammoniæ becomes obviously blue, after a short time. (See *post*, POISONING BY COPPER.)

(8) A few drops of the liquid (A) treated on a watch-glass with a drop of solution of iodide of potassium will give a yellow precipitate, indicating *lead*. This precipitate dissolves in a large quantity of boiling water, but is again deposited on cooling, assuming the appearance of shining scales. The liquid will also yield with a solution of sulphate of soda a white precipitate, either immediately or very soon, if it contains *lead*.

(9) If the liquid saturated with sulphuretted hydrogen throws down a yellow, or yellowish, precipitate, it must be carefully examined. After decantation, it should be washed once only—not with pure water, but with a limpid aqueous solution of sulphuretted hydrogen. If this precipitate consists only of sulphide of *arsenic*, it will completely dissolve in a few drops of liquid ammonia, and be precipitated anew on the addition of an acid. If treated with pure boiling nitric acid, it should disappear, in being converted into arsenic acid; and when introduced into Marsh's apparatus, it should furnish the characteristic bright metallic deposits on a piece of cold white porcelain, and which will immediately disappear on being touched with a solution of hypochlorite of lime or soda. (See *post*, POISONING BY ARSENIC.)

But the yellowish precipitate may turn out to be nothing more than finely-divided sulphur, arising from the decomposition of a portion of the sulphuretted hydrogen under the influence of the air, or of the acid in the liquid (and in case there should be organic matters present, the colored precipitate would be composed of both sulphur and organic matters). If the deposit consists exclusively of sulphur, it will possess none of the preceding characters; moreover, it will undergo fusion at a heat a little above 100° C., and will burn without residue, with a characteristic color and odor.

(10) If the liquid saturated with sulphuretted hydrogen

yields no precipitate, or merely one of sulphur, it should be put into a porcelain capsule, and boiled until the odor of sulphuretted hydrogen has entirely disappeared, and its volume reduced to one-tenth of the original. If now introduced into Marsh's apparatus, it will yield the characteristic spots on porcelain, if it contains *arsenic.*

It will be perceived that, in the analysis just described, arsenic is discovered by two different processes (9 and 10); the explanation is as follows: During the treatment of the suspected organs with concentrated sulphuric and nitric acids, the arsenical compounds present will have passed, either in whole or in part, into the condition of arsenic acid, which is difficult to precipitate with sulphuretted hydrogen. As this precipitation is somewhat capricious, it may happen that arsenic may sometimes be deposited, and sometimes not. Hence the provision of the two processes for detecting this substance.

(11) If the liquid saturated with sulphuretted hydrogen yields no metallic precipitate, nor furnishes any spots when tested with Marsh's apparatus, it is because it contains none of the metals mentioned above; recourse must next be had to the carbonaceous mass left upon the filter: this is to be divided into two parts—(*a*) and (*b*).

The first (*a*) is mixed with some distilled water, and then from half a drachm to a drachm of pure carbonate of soda is added, and the whole boiled for about half an hour; when the whole is thrown upon a paper filter. The mass is repeatedly washed with water, until the disappearance of all alkalinity, and again washed with very dilute nitric acid. The results of these washings being added to the filtrate, the whole is evaporated nearly to dryness on a water-bath. It is then again diluted with distilled water and filtered anew. If it contains *lead,* it will respond to the usual reactions of this metal, especially with iodide of potassium and sulphate of soda.

It is easy to understand why lead should be discovered by two distinct processes. Supposing the animal matters to contain a salt of lead; this would necessarily be converted into the sulphate, in the preliminary action of sulphuric acid. But, as the sulphate of lead is nearly insoluble, a great part of it will remain in the carbonaceous mass upon the filter. It may, however, happen that some of it will be redissolved by the nitric acid employed in the treatment of the carbonaceous mass, and thus be discovered in the filtrate, as above described. Hence, in a medico-legal research, it is proper to provide for each contingency.

(12) If the first portion of the charcoal-mass yields no result, the other portion (*b*) should be treated as follows: the black powder is mixed with distilled water, and from half a drachm to a drachm of pure tartaric acid is added, and the whole boiled for a few minutes. It is then filtered, and the filtrate treated with a Marsh's apparatus. If *antimony* is present, it will yield metallic deposits on porcelain, not disappearing on being touched with the solutions of hypochlorite of lime or soda. (See *post*, POISONING BY ANTIMONY.)

II. *Search for organic poisons. Portion No. II.* (*Table* 2.)—1 and 2.—The second half of the suspected material is divided into small fragments, and diluted with a little distilled water, if necessary, so as to bring it to the condition of thin soup. It is then introduced into a tubulated retort, the beak of which is connected with a porcelain tube.

This porcelain tube passes through a small furnace, and has attached to its extremity a set of Liebig's bulbs containing a solution of nitrate of silver, acidulated with pure nitric acid. Through the tubulure of the retort there passes a bent glass tube, which reaches down to the bottom of the contents; its outer extremity being attached by means of a gum connector to the nozzle of a bellows, by means of which air can be forced into the retort, and the volatile products carried through the porcelain tube. The retort is heated by means of a sand-bath to about 40° C., and a slow and regular draught of air maintained by the bellows. The Liebig's bulbs serve as a regulator: the rapidity with which the bubbles of air pass through them indicating to the operator the proper degree of motion for the bellows. After some time, the operation is stopped in order to ascertain if the nitrate of silver solution is at all clouded. The porcelain tube is now to be gradually heated to redness, and the bellows again put into action.

If, under these conditions, the silver solution does not become clouded, the experiment is brought to an end. If the reverse is the case, it is continued until the precipitate ceases to be thrown down. The contents of the bulbs are poured into a test-glass, and the precipitate is washed several times by decantation. If this precipitate very soon assumes a violet color on exposure to the light, dissolves freely in ammonia, and is insoluble in boiling nitric acid, it is proof that it consists of chloride of silver, and that the chlorine or the hydrochloric acid was derived from the animal matters in the retort; and under the circumstances it is highly probable that it is due to CHLOROFORM (2) contained in these matters. For, since the precipitate could result only from chlorine or hydrochloric acid, these would have been manifested at the commencement of the experiment, *if they had existed in the free state.* But, inasmuch as the precipitate in the silver solution did not occur until after heating the porcelain tube to redness, it is natural to conclude that some chlorine compound, that has no action on nitrate of silver, was decomposed at a red heat into chlorine or hydrochloric acid, which has produced the reaction. Among the volatile substances known in medicine and toxicology, *chloroform* is the only one that will produce this result. In a case of this kind, the characteristic odor of this substance would almost certainly be noticed. (3) If the silver solution in the bulbs does not become clouded, the search for the organic alkalies may be at once proceeded with, according to the process of M. Stas, which will be described farther on. If a strong alkaline volatile liquid, possessing the peculiar pungent odor of tobacco, is the result, it is NICOTINA (4). If solid, a portion of it is introduced into the thigh of a frog (5). If it greatly dilates the pupil, and the residue readily dissolves in water, giving a strong alkaline solution, is precipitated brown by iodine, and easily assumes a nauseous odor, it is ATROPIA (6). If the frog is seized with tetanic convulsions, intermittent in their character, and the residue responds to the characteristic color-test (sulphuric acid and bichromate of potassa), it is STRYCHNIA (7). If the frog exhibits great weakness, and has irregular and intermittent beats of the heart; and if the residue dissolves in warm water, and yields a solution which is not alkaline, but gives a precipitate with tannin; and if the residue itself is colored green by hydrochloric acid, it is DIGITALINE (8). If the frog exhibits complex physiological phenomena, and the residue is crystalline, nearly insoluble in water and ether, soluble in caustic potassa, and responds to the recognized tests for morphia (perchloride of iron, nitric acid, iodic acid), it is OPIUM (9).

Stas' process for separating the alkaloids.—This method is the most reliable of any yet discovered: it is the one originally employed by M. Stas for the separation and identification of *nicotina* in the celebrated Bocarmé case. It is a process that requires the greatest delicacy and care on the part of the operator. It is based upon the following ascertained fact: "All the alkaloids known form with acids, and particularly with tartaric and acetic acid, salts, which are soluble in water and alcohol; and these solutions are easily decomposed by the fixed alkalies. The alkaloids thus set free nevertheless remain in solution for a few moments, and will dissolve in ether if present in sufficient quantity."

The regulated use of water, and of alcohol of different degrees of strength, effects a separation of the foreign matters, and allows us to obtain in a small volume the solution containing the toxic alkaloid.

The organs, or suspected matters, are finely divided, and mixed with about double their weight of pure alcohol at 95°. From fifteen to thirty grains of pure tartaric acid are then added (previously dissolved in a little alcohol), and the whole put into a glass flask, which is heated on a water-bath for half an hour at a temperature of about 70° C. After cooling, it is filtered through paper, the insoluble portion being several times washed with concentrated alcohol and the washings added to the filtrate. The liquid is slowly evaporated at a temperature not exceeding 35° C. The evaporation may be expedited by a current of air, if convenient.

When the greater portion of the alcohol is evaporated, there is left a liquid which throws down fatty and other matters; this should be again filtered through paper previously wetted with distilled water; the filter should be carefully washed afterwards. The aqueous liquid is now carefully evaporated, either in a vacuum or under a receiver containing quicklime or strong sulphuric acid. The solid residue thus obtained is completely exhausted by cold absolute alcohol; this is slowly evaporated either in a current of air or in vacuo. The new residue is dissolved in the smallest possible quantity of cold distilled water, and the solution at once introduced into a long narrow flask of such a size that it will occupy about one-fifth of its capacity. Bicarbonate of potassa in small quantities is put into the liquid, until effervescence ceases. Finally, the flask is very nearly filled with pure ether, and, after a thorough shaking for several minutes, it is allowed to rest. When the supernatant ether has become clear, a small portion of it is carefully removed with a pipette, put into a watch-glass, and allowed to evaporate spontaneously in a dry place. The result is exhibited under two aspects: it is either liquid and volatile, as nicotine; or else it is solid and fixed, like strychnia, or morphia.

(*a*) If the toxic substance is a liquid and volatile alkaloid, the preceding ethereal solution, on evaporation, leaves oily streaks, which slowly collect at the bottom of the capsule. If this latter be slightly heated, a sharp and pungent odor will be perceived, varying according to the nature of the alkaloid.

In order to discover its true character, to the contents of the flask from which the trial test was made are added two cubic centimetres of a solution of caustic potassa (one part to five), and the mixture is shaken up anew. When the ether has completely cleared, it is decanted into a larger flask; the residue is exhausted by three or four additions of ether, and all the ether solutions are united in the larger flask, to which are immediately added two cubic centimetres of water acidulated with a fifth of its weight of pure sulphuric acid. The flask is shaken for some time, and then allowed to rest. The ether is now decanted, and the acid liquid is washed with a new dose of ether. The alkaloid is now in the form of a sulphate, and is no longer soluble in ether, but remains dissolved in the acidulated water. In order to extract it perfectly from this solution, a concentrated aqueous solution of caustic soda is added, and the mixture is exhausted by several successive additions of pure ether. The ethereal solution is allowed to evaporate spontaneously under a receiver containing strong sulphuric acid; the residue will contain the organic alkali in a state of great purity, which will allow its physical and chemical properties to be tested.

(*b*) If the tentative evaporation of the small quantity of ether taken from the flask does not indicate the presence of a liquid alkaloid, a concentrated solution of caustic soda is added to the mixture in the flask, and, after a brisk shaking, the ether is decanted, and more is added, until the mass is completely exhausted. The ethereal liquids, being spontaneously evaporated in a capsule, leave as a residue an aqueous liquid, which holds in suspension some solid matters. If it contains a solid alkaloid, it will have a decided alkaline reaction; and, although in every case it yields a disagreeable odor, this will not be acrid or pungent. In order to isolate the alkaloid from foreign matters, we pour into the capsule some drops of water acidulated with pure sulphuric acid, and, after some time, filter the liquid through paper. The filter should be washed repeatedly with distilled water, and the washings united to the filtrate. This liquid is then allowed to evaporate to one-fourth of its original volume, either in a vacuum, or under a receiver with sulphuric acid or quicklime. Next, a concentrated solution of pure carbonate of potassa is poured upon the residue; and the whole is finally acted upon by absolute alcohol, which redissolves the alkaloid, and deposits it in the crystalline state, after filtration and evaporation. From this, the physical and chemical characters of the substance can easily be determined.

For separating some of the alkaloids, *chloroform* and *amylic alcohol* (especially the former) have been found to be much better solvents than ether. The cases to which these are particularly adapted will be duly pointed out hereafter.

This method, the most exact known, is certainly sufficiently minute, and requires to be employed with all possible carefulness. The results are satisfactory in the majority of cases. The alkaloid is easily concentrated in a small volume, and is freed from the greater proportion of the animal matters with which it was associated. It must, however, be admitted that, in spite of every precaution, it scarcely ever happens that all traces of foreign matters are completely removed; besides, a small quantity of the substance is lost in the various manipulations. Although specially designed for the separation of the alkaloids, it is equally applicable to the discovery of *digitaline*, although this substance is not, properly speaking, a vegetable alkali.

The very complicated process of Stas, above described, may often be much simplified with considerable advantage. For instance, in separating strychnia from organic mixtures, it is often only necessary to digest for some time in water slightly acidified with acetic acid, then strain and filter; concentrate by evaporation; add liquor potassæ in excess, and then shake up with an excess of chloroform. After standing awhile, the chloroform solution of the alkaloid is separated, and allowed to evaporate to dryness, when the deposit is tested in the usual manner (*vide post*, STRYCHNIA).

Dialysis.—The process of dialysis was originally recommended by Mr. Graham, as a very convenient method of separating crystalline bodies from complex organic mixtures. It is essentially the process of endosmose—a moist organic membrane being interposed between two different liquids. The substance found to be best adapted for the septum is *parchment-paper*, which is prepared by soaking unsized paper for a few minutes in a cold mixture of two parts of sulphuric acid and one of water. The *dialyser* is composed of a light hoop of wood, or, better, of sheet gutta-percha about two inches in depth and five or six inches in width, with a piece of moistened parchment-paper stretched over it, and secured by a string or elastic band around it, so as to form a sieve-like vessel. The liquid mixture to be examined is poured into the dialyser, in quantity not exceeding half an inch in depth; this is next floated in a perfectly clean glass or porcelain basin containing distilled water, in quantity about four or five times that of the liquid in the dialyser. Any crystalline matters (termed *crystalloids* by Graham) contained in the latter will begin at once to pass through the septum into the clear water on the other side; while the non-crystallizable substances (termed *colloids*) will remain behind. In the course of twenty-four hours the process of diffusion will be completed. The outer liquid, or *diffusate*, should be evaporated on a water-bath to a small volume, or to dryness, and the residue, if sufficiently pure, examined by the proper reagents. A drop or two of the concentrated liquid may be evaporated on a piece of glass, and the crystals examined by the microscope.

This process answers very well where the quantity of the suspected (crystalloid) body is considerable: it is especially adapted for the examination of the mineral poisons, as arsenic, corrosive sublimate, tartar emetic, etc. In identifying the two latter poisons, its use is very appropriate as a preliminary test, inasmuch as by it we can separate from the contents of the stomach the identical substance that has caused death, whereas by the ordinary chemical processes this cannot be done, but merely the metallic bases of the salts are obtained—viz., mercury and antimony. The process of dialysis has not been found so well adapted to the separation of the *organic* crystalloids (alkaloids); nor, in general, where the amount of the suspected material is small. In both the latter cases, the diffusate is very apt to be contaminated with impurities (colloids) which have passed through the septum, rendering a further purification necessary.

CHAPTER VIII.

MEDICO-LEGAL QUESTIONS CONNECTED WITH POISONING.

THE matters considered in the preceding chapters embrace nearly all the medico-legal points connected with the subject of poisoning. It may, however, be profitable to the student to group these questions together in their proper order and importance, so as to present a clear and definite picture of the subject. These medico-legal questions will naturally present themselves in every case of poisoning that comes up for trial. Hence it is desirable that the expert should be thoroughly instructed in relation to them, so that, in his evidence, he may be able fully to sustain his positions. As many of these questions presuppose a knowledge of medicine as well as of chemistry, it will be evident that either the expert should unite in himself this twofold character, or else that two experts—a chemist and a physician—should be associated in the case. The following are the most important questions that will commonly be asked at a trial of a poisoning-case:

1. *Is the death or sickness to be ascribed to poison?*—This question is fundamental: it underlies all the others, since it compels the expert to submit to the court and jury the *proofs* of the alleged poisoning. It matters not whether the case has proved fatal or not, justice demands that science should bring forward positive proofs of the alleged crime.

We have already considered these proofs in detail (*ante*, p. 45). They embrace, (1) the symptoms; (2) those furnished by the post-mortem signs; (3) those afforded by chemical analysis; (4) those derived from experiments on living animals; to which may be added, (5) the circumstantial proofs. We have seen how impossible it is in any case to determine the question of poison either by the symptoms alone (since there are no symptoms *exclusively* characteristic of any poison), or by the pathological lesions alone, or even by both together (*ante*, p. 68). But it should not be forgotten that it is nearly always in the power of the expert (or of the medical man in attendance) to discover the poison in the matters vomited or purged, in the urine, and in stains upon the clothing, or on articles of furniture. In non-fatal cases especially, the neglect upon the part of the medical attendant, who may suspect the administration of poison, to make these important examinations of the food and excreta, and his reliance merely on certain *symptoms* which the patient has exhibited, in order to sustain a charge of poisoning, cannot be too severely reprobated.

2. *What is the nature of the poison that has caused the sickness or death?*—It has already been shown (p. 67) that in a case of

poisoning the law always demands "satisfactory proof" of the fact: it does not prescribe the *nature* of this proof. It has been held by some, that to establish the legal proof of poisoning, the *identical poison* that caused the death should be obtained from the body, or other material, and be produced as a *corpus delicti.* This doctrine is, however, not tenable: as well, it might be urged in reply, might it be required in a case of homicide by a blow upon the head, and where a fractured skull gave unequivocal evidence of the cause of death, that the weapon of the murderer should be produced as the *corpus delicti.*

As regards poisoning, the rule to be observed in connection with the chemical proof is, *that whenever it is possible* the analyst should recover the identical poison alleged to have been taken, and exhibit it in court. But this is not always possible, as in the case of some of the vegetable and animal poisons which undergo decomposition in the body: in such cases we must be satisfied to exhibit all the known and admitted chemical tests, together with (in certain cases) the *physiological* proofs (see *ante*, p. 80). But in the case of the mineral poisons it is possible, in many instances, by means of *dialysis* (see p. 113), actually to recover the identical poison employed—*e.g.* arsenious acid, tartar emetic, corrosive sublimate, sulphate of copper, etc.; and, further, in each case to obtain the *metal* in a state of purity; and from this to demonstrate satisfactorily all the recognized chemical reactions of the poisonous mineral.

When all the combinations of a poisonous metal are equally dangerous, it is not essential to the cause of justice that the poison should be detected *under the precise combination in which it was administered*, provided the metallic base is discovered. Thus, in poisoning from either arsenic, tartar emetic, or corrosive sublimate (although possible for the analyst, as shown above, to produce these identical substances), it is regarded as sufficient if he can satisfactorily determine the presence of the *metallic bases* of these poisons, —viz., arsenic, antimony, and mercury.

3. *Was the substance administered capable of causing death?*—This question is likely to arise only in non-fatal cases. If it

can be shown that the substance given with the intention of producing death was really not of a poisonous character (although supposed to be so), conviction would not follow. So also, if the substance were poisonous in large doses, *e.g.* oxalic acid, and only a small quantity—a few grains—had been administered, it would not come within the statute. (See Taylor, On Poisons, p. 183.)

4. *Was the poison taken in sufficient quantity to cause death?*—In many instances the discovery of a large amount of the poison in the body leaves no doubt of the true cause of death. But the expert must beware of falling into the error of supposing that the finding of only a minute quantity of the poison necessarily negatives the charge. It has been satisfactorily shown (see *ante*, p. 70) that in some cases of fatal poisoning not a trace of the noxious substance can be found by the analyst after death. Full and sufficient reasons were given for this failure to detect the poison. It should never be forgotten that the quantity of poison extracted from the body by analysis does not represent the quantity taken. Of course, the discovery of only a minute quantity of the poison (especially of a substance employed in medicine) will always suggest the possibility of its having been taken medicinally, unless the other elements of proof are sufficiently strong. (See *ante*, p. 59.)

5. *When was the poison taken?*—The settlement of this question is of importance both to the prosecution and the defense. As a general rule, the symptoms come on soon after the poison is swallowed; but the exact time varies for different poisons and for different conditions of the system (see *ante*, p. 48). In the case of poisons administered in small and repeated doses (slow poisons), this question is more difficult to determine; although each fresh accession of symptoms may be regarded as indicative of a fresh dose of the poison. It should be remembered also that, in some cases of fatal poisoning, a true intermission of symptoms occurs, which may lead to a serious error in regard to the time of the administration of the poison.

6. *Is it possible for the poison to have completely disappeared from the body, without leaving any trace of its presence? and in*

what time?—This question may be answered affirmatively both as regards the living and the dead body. Thus, violent vomiting and purging may remove the whole of it except the small quantity absorbed. And if the individual survive long enough for elimination (sixteen days for arsenic, see *ante*, p. 28), not a trace of it may be discovered after death. In the living body, the analysis of the urine forms a very exact index of the presence of the absorbed poison in the system: this is especially true of mineral poisons.

7. *Could the poison extracted from the body be ascribed to any other source than to poisoning?* — The presence of poison, especially in considerable quantity, in a body is *primâ facie* evidence of poisoning; but it is not, *of itself*, positive proof (see *ante*, p. 68). If found in very small quantity, this may be ascribed to medicinal administration (in the absence of the other proofs of poisoning). Again, it may be traced to accidental contamination; an instance of which is given by Tardieu and Roussin (*loc. cit.*, p. 136), where the lining membrane of a stomach was found smeared over with the oxide and carbonate of copper, which was ascertained to be solely due to the presence of a large pin, that had accidentally fallen into the jar, after the autopsy.

Again, the impurities contained in the reagents of the chemist, if he is not sufficiently careful, may account for the accidental introduction of certain poisons, as arsenic, antimony, lead, etc. Finally, it has been supposed that the natural decomposition of the human body would give rise to certain products which, if not actually identical with, closely resemble, some of the organic poisons. This has been alleged to be the case, at times, with prussic acid. But the allegation lacks proof. This question will be discussed more fully under PRUSSIC ACID (*post*).

8. *Was the poisoning the result of homicide, suicide, or accident?* —This is a question for the jury to determine, rather than for the expert. Nevertheless, in some cases it will be in his power to throw light upon it, as in the instance of the *corrosives*, where the evidence of resistance on the part of the deceased is shown by the spilling of the fluid upon the face, neck, and chest of the individual, together with other

circumstantial evidence of a similar character, all indicating homicide.

9. *Can poisoning be pretended?*—There can be no doubt that, as many diseases are simulated for sinister purposes, so an individual may pretend that he has been poisoned, and even exhibit poisonous mixtures alleged to have been vomited, as proofs of his statement. Like any other impostor of this description, such a person must be carefully watched: this will generally lead to his detection. The idea of being poisoned is a very common delusion of the insane.

The above medico-legal questions have been taken chiefly from the treatise of Tardieu and Roussin, to which the reader is referred for more extended details.

CHAPTER IX.

DUTIES AND PRIVILEGES OF MEDICAL EXPERTS.

THE subject of *expert evidence* is one of the greatest importance, especially in criminal trials for poisoning. Here, indeed, the final issue often depends materially upon the character of the expert testimony. It becomes, then, a question of the utmost consequence to determine what are the proper duties and privileges of medical experts.

The term "expert witness" literally signifies a *skilled* witness—one who has accurate knowledge of the matter under consideration. Such witnesses are "chosen on account of their special knowledge or skill in particular matters, to testify or make a report embodying their opinions" (Elwell). The "expert" witness does not testify to *facts*, like the ordinary witness; but he gives his *opinion*, based upon facts that have been testified to by others. It is his special function carefully to weigh all these facts, to sit in calm judgment upon them, and to deduce conclusions from them, which he delivers to the court and jury as his *opinions*.

For the *consequences* of his opinions he should clearly understand that he is in no wise responsible. He is bound by the solemnity of his oath to tell all and to suppress nothing of what he conscientiously believes to be *the truth*, no matter what may be the result to the accused. In the language of Dr. Percival, "he should use his best endeavors that his mind be clear and collected, unawed by fear, and uninfluenced by favor or enmity."

How important, then, that the individual, who assumes the functions of an "expert" witness in a criminal trial, should so clearly understand the matter on which he is to testify, and be so familiar with its every detail, as to preclude all possibility of error on his part! Yet it is notorious that nearly every criminal trial in our country is hampered, if not disgraced, by what has been sneeringly termed by the newspapers the "war of the experts." So much is this the case, that the public have come to expect this collision as a matter of course; and, as a consequence, they are inclined to reject the whole expert testimony as entirely superfluous, if not positively worthless,—a result which, it is to be feared, is not unfrequently reached also by the jury, to the great and manifest disparagement of justice.

In trials for poisoning, it is by no means unusual to find medical men summoned as "experts," both by the prosecution and the defense, who have never made the subject of toxicology a special study, and who must therefore be ignorant of the important details of this science; but who, nevertheless, because they are *doctors*, and are erroneously supposed to know, will venture to assume this most responsible function, and will even presume, from the witness-stand, to enlighten the court and jury on one of the most intricate branches of science, and will hazard "opinions" which may probably determine the momentous issues of life and death! If it were only possible to exclude these improvised experts from poison-trials, and confide the responsibility to men of known and recognized toxicological ability, there would occur far fewer occasions in which this conflict of expert testimony would be witnessed; for it is to be observed, that among *genuine* experts—persons of purely scientific attainments, uninfluenced by either prejudice or favor, not mere partisans—this difference of opinion on a purely professional subject is much less likely to occur. Accustomed to observe the facts as presented, from the same scientific stand-point, and to apply the same chemical tests in order to determine certain results, there would be a far greater likelihood of their arriving at similar conclusions.

We have heard it gravely asserted from the bench, in a certain criminal trial for homicide, in the charge to the jury, that "one expert is just as good as another." This doctrine we regard as both unsound and pernicious. If the expert opinion of A, a thoroughly educated toxicologist, is to be neutralized by that of B, who is merely a practitioner of medicine, who does not see what serious danger might result to the cause of justice and of human life, if the jury is to estimate the "opinion" of both as of equal importance? It is easy to understand what might be expected from a jury, instructed as above mentioned from the bench, and bewildered and deceived as to the relative value of the "expert" testimony, by a crafty and unscrupulous counsel.

The question of the proper remedy to be applied to this faulty system of expert testimony has engaged the attention of many able minds. Dr. Wharton says (Wharton and Stillé's Med. Jurisp., 1873, ii. p. 1248), "The radical defect, however, of our present Anglo-American practice, in this respect, is the volunteer position of experts, which makes them, to a large measure, the mouth-piece of a party who often only selects them because their pre-ascertained views suit his purposes; or who only presents them with such materials as subserve his interests. In what way this defect can be removed is one of the most important questions to which social science can now be addressed." In Great Britain and France this arbitrary voluntaryism still prevails. In France, the judge may call in experts according to his discretion; he is sometimes guided by his own partiality, and sometimes by the popular reputation of some physician, without any assurance that the expert thus called has made the particular subject-matter of the trial his specialty.

In Germany it is, fortunately, otherwise. In criminal cases, the experts first summoned are exclusively those whom the

State, after proper examination of their competency and skill in such particular inquiries, has duly authorized to act for this purpose; while in addition to this, there is organized a tribunal of experts, to which the opinions of expert witnesses can be referred (Casper's Gericht. Med., Berlin, 1871, i. § 3).

Under the present head some allusion may be made to the duties of "medical experts" when employed to make examinations of a body alleged to have been poisoned. The following extract from the late edition of Wharton and Stillé's Medical Jurisprudence, 1873, ii. p. 1246, presents very correctly our own ideas in relation to this subject:

"*Examinations made ex parte, when there could have been notice to the other side, are inadmissible.*—Examinations, for instance, of an alleged lunatic, conducted by a professed specialist, or examination of blood on clothing, or of alleged poison contained in the stomach of a deceased person, or in bottles or utensils, can in most cases as readily be made upon notice to the opposing interest, as without notice. For various reasons, such notice, if practicable, should be given. First, it is a familiar principle of law that depositions purely *ex parte* are inadmissible: such testimony being liable to be affected by fraud or prejudice, and from want of cross-examination being necessarily imperfect. Secondly, there are peculiar reasons why *ex parte* examinations of the character here noticed should be undertaken only upon notice to the opposing interest. In such examinations everything depends upon the accuracy of the tests employed; the exhaustiveness of the exploration; the fidelity and cautiousness of the examiner. In questions of poison and of blood-stains, in particular, it is important that there should be on the spot, at the time of the examination, the representative of the adverse interest, for the purpose of seeing that the objects examined had not been previously tampered with; that no foreign elements were interposed; that the investigation was conducted with scrupulous conscientiousness." . . . "But when, after these preliminary inquiries (the coroner's inquest) are over, an examination is desired by one of the parties in interest, and when this examination relates to a subject-matter not fleeting, but continuing, then the examination is analogous to the deposition of a witness, and the policy of the law requires that it should be taken only after notice to the other side."

"Sometimes, perhaps, testimony of value inadvertently taken, will be excluded by the application of this rule; but this will be abundantly compensated for by the suppression of those inquisitorial and imperfect investigations by which the administration of public justice has been so much disgraced; and by the investing of expert testimony with checks and sanctions by which alone can its dignity be restored."

The violation of the above equitable principle was very forcibly illustrated in the first Wharton trial at Annapolis, Md., in 1872. In this case, the body of General Ketchum, who was alleged to have been poisoned with tartar emetic, was *thrice* examined, and each time *exclusively* by the State's experts; having been also twice exhumed for that purpose, —the last time *secretly*, by order of the court, during the progress of the trial, without even the knowledge of the prisoner's counsel or her expert witnesses! (See Review of this trial, by the author, in Am. Jour. of Med. Sci., April, 1872; also Wharton and Stillé's Med. Jurisp., *loc. cit.*)

Privileges of experts.—Subpœnas.—Compensation.—The question has frequently been raised whether an "expert" witness is obliged to obey the process of a subpœna, like any ordinary witness, and testify in a given case *as to his opinion*, without a previous guarantee of an adequate pecuniary compensation. This is a matter that especially concerns *medical* experts, inasmuch as in cases of medical jurisprudence, and particularly in poison-cases, they are so frequently summoned, and the final result so much depends upon their testimony. In an important trial for poisoning, the issues of life and death often hang suspended upon the "opinion" of the expert, as given in his testimony. In order to be able to give this opinion conscientiously, the witness should be personally present throughout the trial, however lengthy this may be; he must listen attentively to all the evidence; he must be in close and intimate conference with the counsel, instructing them in various points of a professional and scientific nature, suggesting proper questions to be put to the professional witnesses, pointing out the blunders of careless or unqualified chemists and physicians who may have been employed in the case, and often performing experiments in order to verify his positions. Now, to do all this requires the expert to absent himself for a length of time from home and from his daily professional duties, to the manifest detriment of his business. We speak not here of the additional labor, anxiety, and responsibility involved where the expert has been required to make the chemical analysis of the body, in a case of supposed poison.

The question, then, presents itself with much force—how is the expert witness to be compensated for his time and services? Has the State the right to compel him to attend and testify at the trial? Has the defense this right also? Let us examine these questions.

In the first place, it is perfectly clear that an expert witness cannot be subpœnaed to attend a trial *out of his own State.* If he goes at all, it must be voluntarily; and he is at liberty, of course, to arrange his own terms; these would naturally be regulated according to the circumstances of the case. Should, however, the trial occur within the limits of his own State, the matter assumes a different form. Every person is compelled by law to obey the mandate of a subpœna, within his own State: the medical expert is no exception to this general rule. Such seems to be the naked law upon this question. High authorities, however, have sometimes ruled differently. Lord Campbell's opinion in Betts *v.* Clifford (Warwick Lent Assizes, 1858) was that a scientific witness was not bound to attend on being served with a subpœna, and that he ought not to be subpœnaed. If the witness knew any question of *fact*, he might be compelled to attend, but he could not be compelled to give his attendance to speak to matters of *opinion.* In the case of Webb *v.* Page (Car. and Kir. Reports, p. 23), the late Mr. Justice Maule ruled as follows: "There is a distinction," said his lordship, "between the case of a man who sees a fact and is called to prove it in a court of justice, and that of a man who is selected by a party to give his opinion on a matter on which he is peculiarly conversant from the nature of his employment in life. The former is bound, as a matter of public duty, to speak to a *fact* which happens to have fallen within his own knowledge; without such testimony the cause of justice must be stopped. *The latter is under no such obligation;* there is no such necessity for his evidence, and the party who selects him must pay him." In the case referred to by Mr. Justice Maule, a skilled witness had been subpœnaed, but he refused to give evidence unless first paid for his services and loss of time. (Med. Times and Gaz., April 26, 1862, p. 432.) A barrister quoting this ruling goes on to say: "There is one reason why I should not advise any person in the position of a skilled witness, totally to disregard a subpœna. It is quite clear that should such a person fail to attend a trial, no attachment could ensue, even if he were called, as is usual, upon the subpœna, because the party subpœnaing him could not make the requisite affidavits that he was damnified by the witness's absence, and in what respect. But such party might bring an action for damages; and although he would recover none, he might not only worry, but might even put the defendant to considerable expense. Although, therefore, I could not advise a total neglect of the subpœna, the safest course would be to obey it, and demand expenses before giving evidence. Such expenses would be only those allowed for a professional witness (not special fees). To permit him legally to demand a high fee, would perhaps look somewhat like legally countenancing a bribe."

Dr. Taylor, from whose work on Medical Jurisprudence (Am. ed., 1873, p. 40) the above is quoted, remarks, "that Lord Campbell's dictum in reference to the distinction between *fact* and *opinion* confers no practical benefit on witnesses. It is at all times difficult in science, and in the medical sciences particularly, to separate them; and if a man appears to testify to a medical or scientific fact, he cannot avoid giving an opinion arising out of the fact."

Granting all the force of the above opinions, it can hardly be supposed that, in an important trial for murder by poisoning, the counsel for either the prosecution or the defense would hazard the issue upon the testimony of a *reluctant* expert, who has been dragged perhaps hundreds of miles from his home and professional business, and has been detained for days or weeks, and then insulted with the proffer of the paltry allowance of an ordinary witness. Although the law might compel him to go upon the witness-stand, and to be sworn to state "the truth, the whole truth, and nothing but the truth," who does not see that, while the witness may not violate the letter of the law, his testimony may be practically neutralized by the *manner* in which he gives it? For, after all, the moral power of the expert, in such cases, is exhibited not so much by his mere answers to categorical questions, as *indirectly*, by the aid afforded to his counsel in a multitude of ways. He may, for instance, refuse to hold any conference with counsel on the delicate points of the case; he may decline to suggest any valuable hints as to the manner of putting questions to the witnesses on scientific subjects; he may abstain from performing experiments either for the purpose of confirming or of rebutting certain points of the evidence: in fine, he may take such a *passive* part as an "expert" witness, that he will prove rather a detriment than a help to the party summoning him. From some experience on this subject, we would strongly advise against the attempt to *compel* a skilled witness to testify in a trial, without a previous satisfactory understanding as to his adequate compensation.

Let it not be thought that we take too mercenary a view of this matter. On what principle of justice or morality, we would ask, should a professional witness be refused an adequate compensation for services which have cost him years of study and labor, as well as no inconsiderable outlay of money? We desire to speak very plainly and pointedly on this subject: it is one too little noticed and insisted on in works of this character. According to good authority, it will always be proper for the professional expert to ask the court for a proper compensation before he gives his evidence. *Generally*, some compensation will be allowed by the court, additional to the ordinary witness-fee; but this extra allowance is likely to be altogether inadequate to the services rendered. In fact, the whole matter seems to depend very much upon the generosity of the court, and upon the supposed importance of the witness summoned in the case.

Another point to be noticed is that, where a toxicologist has been employed, either by the State, or by a private party, to make a chemical analysis, in a case of suspected poisoning, he should invariably stipulate beforehand about the payment of his fees; otherwise, he will very likely be forgotten or totally ignored after the trial is over. He should remember that no subpœna can compel him to render this service. It is perfectly voluntary on his part. He may decline it altogether, if he so pleases. If he agrees to undertake it, he is at liberty to state his own terms. But, as the result of a somewhat extended experience, we would caution him as to *how* he makes his agreement. For example, it is a very common practice in this country, in a case of suspected poisoning, after an inquest has been held, for the district attorney of the county to send the viscera supposed to contain the poison to some reliable chemist, accompanied by a polite request to him to perform the analysis with the utmost possible expedition, and adding, that an adequate (or perhaps it is worded, *reasonable*) compensation will be paid him by the county for his services. Probably this letter will be accompanied by another from some medical man, who has performed the post-mortem examination, and who has recommended his chemical friend to the authorities, as the proper person to make the analysis. Let him be cautious how he becomes a party in this transaction. Without any *intention* on the part of any one to wrong him, he will almost certainly *be wronged*, unless he is very careful how he acts. Suppose him to be young in the business: he enters upon the work ardently and diligently; he devotes probably a week or two to the analysis; the result is communicated to the law-officer in due form; he goes to court, and spends another week or two at the trial, where he delivers his evidence—itself, no trifle; on this evidence mainly will depend the conviction or the acquittal of the prisoner—his life, or his death! After the trial is over, he is probably congratulated on his skill and ability as a toxicologist; but when the question of *payment* comes up, difficulties thickly beset his path. His friend the district attorney has no authority, or funds at his disposal, to pay him; the county commissioners, or some analogous board, must first meet, and *vote* his compensation. From the material usually composing such "boards," who would expect that a fair or proper appreciation would be put upon his really valuable and scientific services? They will, in all probability, vote him a sum about equal to what they pay for some petty mechanic's work done for the corporation—possibly about the fifth, or the tenth, of what he is justly entitled to; and, sad for the poor expert, *there is no redress!*

The above is no mere fancy sketch: it has its counterpart, we venture to say, in the experience of nearly every toxicologist in our land. What, then, should he do to secure himself against such imposition? His only remedy is *to insist in advance on a bond duly signed by all the commissioners, or by some equally responsible party, for the payment of the fee agreed upon.* However mercenary such a course may appear to the uninitiated, we fearlessly recommend it, after some personal experience, and also as, in the opinion of many leading toxicologists, the only safe one for the professional expert, either for his own protection, or for the protection of his profession. Even with such a safeguard it has happened to the author, on one occasion, to be actually compelled to sue "the commissioners" of a certain county in Pennsylvania, in order to recover a fee for the performance of a chemical analysis and giving evidence in court, which they had previously solemnly bound themselves to pay! In another case, where the court in a neighboring State had ordered a toxicological examination to be made, and had agreed with us for the compensation, we were compelled to incur the expense of a lawsuit against the county, and to wait for more than a year before the recovery of our fee.

A case occurred in a certain county in Pennsylvania, a year or two ago, which forcibly illustrates this anomalous state of affairs. A woman was arrested and thrown into prison, on suspicion of having poisoned another with arsenic. The body of the deceased was examined by a medical friend of the author, and the viscera were sent to Philadelphia with a request for us to make the chemical analysis, and further stating that the authorities would pay all necessary expenses. Warned by previous experience, an answer was returned stating that, before commencing the examination, it would be necessary for the county commissioners to give a joint bond for the payment of the proper fees. After various quibbles on the part of the authorities, endeavoring to bring about a change in our decision, though ineffectually, we put an end to the ridiculous and disgraceful business (which occupied fully six weeks) by returning the package unopened, by express; the other party taking the risk of loss and breakage by public conveyance! During all this time a *possibly innocent* woman was immured in a prison, under the dreadful charge of murder; and all simply because of the parsimony of the county officers! We conceive that a grievous wrong was here perpetrated, both upon the person merely *suspected* of guilt, by an unnecessary detention in custody, and likewise upon the cause of justice; since, if the prisoner was guilty, the State incurred the risk of being unable to prove the guilt by the chemical evidence, in consequence of the long delay, and still more by its loose way of dealing with the viscera supposed to contain the poison, in exposing them to the hazard of ordinary transportation. The final issue of this singular case is unknown to us.

An expert witness for the defense may sometimes be subjected to a similar fraudulent deprivation of his just dues. A single instance only will here be given. About two years ago a case was tried in a neighboring county where a man was indicted for poisoning his wife with arsenic. A small quantity of this substance was discovered by the analyst, in the body of the deceased. Prof. R. E. Rogers and ourselves were engaged by the counsel for the defense as expert witnesses to rebut the charge of poisoning, by showing (as was clearly proved) that the amount of absorbed arsenic discovered in the body of the deceased might be satisfactorily accounted for, from the fact of her having taken this substance as a medicine, by the advice of her physician (who so testified), for some time previous to her death. The prisoner was acquitted,—his acquittal being unquestionably the result of the testimony of his expert witnesses, who were dismissed by the counsel with a profusion of thanks, and promises of speedy remuneration, after being detained at the trial fully ten days, at great personal and professional loss. The remainder of the story must be told, although it reflects severely against a member of the bar,—one of the prisoner's counsel. Previous to the trial, this individual, together with his colleague, waited upon us for the purpose of engaging our services as experts, and it was represented that we should be adequately remunerated; but at the same time a subpœna was served, which we were, of course, compelled to obey. Naturally supposing that we were to be dealt with in good faith, we both gave our unremitting attention to the case. Although it is well known that the individual above alluded to received abundant means from the prisoner and his friends for the express purpose of paying his expert witnesses, he has ignored the whole matter, taking refuge under the wretched pretext that no *legal* agreement had been entered into between the complainants and himself! It is to be hoped that our professional brethren will take warning against similar fraud and imposition.

Another subject of annoyance, and occasionally of positive grievance, to the "expert" witness, is the rude and defiant tone assumed by a certain class of lawyers in the cross-examination. Although it is commonly understood that no gentleman would indulge in coarse and boisterous bravado while examining a scientific witness, yet it does occasionally happen that a barrister is betrayed into it. There is no doubt that the law intrusts almost unlimited powers of interrogation to counsel, for the purpose of eliciting the truth; but, nevertheless, there are bounds beyond which he should not venture. In the language of Chief-Justice Erle, alluding to an imputation having been cast by counsel upon a skilled witness for truthfulness: "The freedom of question allowed to the bar was a public nuisance, and the barrister who made such an imputation ought to be prosecuted." "In his experience, he had seen counsel so abuse their privilege, that he had cordially wished a power could be instituted that they might be prosecuted for a misdemeanor." The same spirit which betrays the lawyer into the fault just mentioned, will further lead him in his "forcible" address to the jury to misrepresent and distort medical facts, in a manner wholly irreconcilable with truth.

Prof. Taylor truly remarks (Med. Jurisp., Am. ed., 1873, p. 50): "The treatment of a medical witness, in passing through the ordeal of an examination at a criminal trial, will depend very much upon the class of counsel who is opposed to him. Assuming that he is properly prepared for the discharge of his duties, and that the questions put to him are answered fairly and truly, according to his knowledge and experience, without exaggeration or concealment, he has no reason to fear any attempt at intimidation. Barristers, for the most part, know that by this line of conduct they lose more with the jury than they gain by the attempt to confuse the witness." "A public writer in commenting on this subject says: 'But the hardest and most unfair part of the system (of cross-examination) is when witnesses have to bear a loud and insulting tone or gesture, without remonstrance or retaliation. A counsel may very plainly imply that a respectable witness is a person of doubtful character and not to be believed on oath, or that he is ignorant, and a bungler in his profession ; but if the witness retorts that the barrister's eloquence and sympathies are hired, or if he gives vent to any other words of retaliation in his natural indignation, the court is against him.' Whatever may be the importance of a case to a prisoner, nothing can justify the putting of questions in a loud and insulting tone to a skilled professional witness." Those who were present at the two celebrated Wharton trials at Annapolis, Md., in 1872 and 1873, will readily recall a counterpart of the above description of the barrister of "a loud and insulting tone."

Prof. Taylor (*loc. cit.*, p. 51), in further reprobating this irritating and uncourteous manner of examining the witness, remarks: "It may be that criminal cases fall more into the hands of the second class of barristers to whom Mr. Stephen alludes,—namely, those who disgrace a noble profession."

The reader will do well to consult Dr. Elwell's medico-legal treatise on "Malpractice and Medical Evidence," also

Dr. Taylor's large work on "Principles and Practice of Medical Jurisprudence," 1873, for fuller details in reference to important points in connection with Medical Evidence.

CHAPTER X.

CLASSIFICATION OF POISONS.

Among the numerous classifications of Poisons that have been proposed at different times, two only need claim attention. One of these is founded on the source, or natural kingdom from which the poison is derived, and is expressed by the two classes of Inorganic and Organic Poisons; and also by those of Mineral, Vegetable, and Animal Poisons. The other classification, which may be termed the *physiological*, has reference to the effects of poisons upon the healthy animal system. To this latter we give the preference, as being most in accordance with practical usefulness. This method of classification was originally proposed by Fodéré, and adopted by Orfila. It divides poisons into four groups: Irritants, Narcotics, Narcotico-acrids, and Septics or Putrefactives. This arrangement has been substantially followed, with certain modifications, by most modern authorities. The principle on which it is based is undoubtedly the correct one, viz., *the mode in which poisons affect the human system in its normal or healthy state.* Like any other system proposed, this classification is open to some objections: thus, it necessarily separates from one another substances derived from the same natural kingdom. But this objection has but little practical weight, and is more than counterbalanced by the advantages gained. Tardieu, following out the above principle, divides poisons into five classes: 1, Irritants and Corrosives; 2, Hyposthenisants; 3, Stupefacients; 4, Narcotics; and 5, Nervosthenics. Dr. Taylor's division is into two classes: 1, Irritants; 2, Neurotics; the latter being subdivided into (*a*) Cerebral, (*b*) Spinal, and (*c*) Cerebro-spinal. Professor Guy's classification is "a convenient compromise between the claims of physiology and natural history." He makes two great divisions: 1, Inorganic; 2, Organic Poisons. The Inorganic are subdivided into (*a*) Corrosive, and (*b*) Irritant. The Organic into (*a*) Irritant; (*b*) Affecting the brain, (*c*) Affecting the spinal cord, (*d*) Affecting the heart, (*e*) Affecting the lungs.

The classification adopted in the present treatise is that of Dr. Taylor, with a few modifications, as being the most simple, and, at the same time, sufficiently comprehensive.

I. IRRITANTS.—This class of poisons includes all those substances whose action is exerted especially upon the mucous membrane of the alimentary canal. Their effects are generally sufficiently well marked: these are an acrid and burning taste on swallowing, nausea, vomiting, purging, great pain in the abdomen, increased by pressure, cramps of the stomach; the matters vomited and purged being frequently mingled with blood. In fatal cases, the autopsy reveals marks of great irritation and inflammation, and, as a result of the latter, ulceration, perforation, and gangrene.

The Irritants may be subdivided into two orders: 1, Simple Irritants; and 2, Irritants possessing remote specific properties. They may further be separated into three sections, depending on the source from which they are procured, viz., Mineral, Vegetable, and Animal; and the Mineral are again subdivided into (*a*) Non-metallic, and (*b*) Metallic poisons. Some of the irritant poisons possess *corrosive* properties—destroying the tissues with which they come in contact, by virtue of chemical affinities. Examples of this are afforded in the mineral acids, the caustic alkalies, corrosive sublimate, etc. The corrosives, as a rule, manifest their action *immediately;* the other irritants more slowly. Although an irritant may never act as a corrosive, a corrosive will always act as a simple irritant, if diluted.

II. NEUROTICS.—The second division of poisons includes those whose action is especially directed to the great nervous centres—the brain and spinal cord. The symptoms manifested are totally distinct from those usually occasioned by Irritants. They consist of drowsiness, headache, giddiness, delirium, stupor, and sometimes convulsions. In some exceptional instances, an irritant impression seems likewise to be produced on the alimentary canal. A natural subdivision of this class is into—1, *Cerebral;* 2, *Spinal;* and 3, *Cerebro-spinal.* The first of these comprises (*a*) the well-known *Narcotics*, of which opium is the type, and (*b*) the Anæsthetics; the second (Spinal) includes those which act primarily and specially upon the spinal cord,—of which strychnia is a notable example; the third (Cerebro-spinal) includes such as influence both brain and spinal cord, producing delirium, coma, convulsions, and paralysis,—of which conia, aconitina, and atropia are examples. The *cerebro-spinal* order of Neurotics embraces by far the largest proportion of this second class of poisons. For facility of description and arrangement, they may be grouped as follows: 1, *Deliriants;* 2, *Depressants;* 3, *Asthenics*, or those which occasion death by *shock.* The above arrangement is to a great extent an arbitrary one, and is, of course, necessarily imperfect.

As already stated, the boundary-line between these classes of poisons cannot always be clearly drawn. For while some of the irritants will occasionally produce symptoms that would more naturally be expected from the neurotics—such as paralysis, convulsions, delirium, and coma,—so, on the other hand, the neurotics may at times be attended by the symptoms of an irritant poison. It is, of course, very important to bear these facts in mind, in diagnosticating any particular case of poisoning.

TABLE OF CLASSIFICATION.

CLASS I. IRRITANTS.	Order 1.	IRRITANTS PROPER.	MINERAL.	Non-metallic. Metallic.
	Order 2.	IRRITANTS PRODUCING REMOTE SPECIFIC EFFECTS.	VEGETABLE. ANIMAL.	
CLASS II. NEUROTICS.	Order 1.	CEREBRAL.	Narcotics. Anæsthetics.	
	Order 2.	SPINAL, OR TETANICS.		
	Order 3.	CEREBRO-SPINAL.	Deliriants. Depressants. Asthenics.	

CHAPTER XI.

CLASS I.

IRRITANT POISONS.

IRRITANT POISONS are here understood to include those whose action is chiefly, if not exclusively, exerted upon the mucous membrane of the alimentary canal, causing an irritation more or less violent, which often amounts to the most decided inflammation, to corrosion, and even to complete destruction of the parts with which they come in contact. The term *corrosive* is applied to such substances as occasion the more violent effects last mentioned. Nearly all the ordinary irritant poisons belong to the inorganic kingdom; a few are found among organic bodies, being chiefly of vegetable origin, as the *drastics*.

Whilst most of the poisons classed together under the head of irritants appear to produce their effects solely in a local manner, *i.e.* by setting up an inflammation and its consequences in the stomach and bowels, there are some which, in addition to this, do undoubtedly produce a remote specific effect, that seems especially directed to the nervous centres, occasioning symptoms not explicable by their merely local action. Examples of these are afforded in arsenic, tartar emetic, salts of mercury, copper, oxalic acid, etc. To this latter subdivision Tardieu assigns the name of *hyposthenisants*. Their peculiarities will be noticed under their respective heads. Irritants may very properly be considered under the two subdivisions of (1) Irritants proper, and (2) Irritants occasioning remote specific effects.

Common symptoms.—A pungent, hot, or even burning taste in the mouth, sometimes metallic; a sense of burning in the throat, extending to the stomach; pain, more or less violent, in the stomach, often extending over the abdomen, which is tender on pressure; almost always, violent vomiting, accompanied with nausea and retching, occurs very early, the matters vomited being often mixed with glairy mucus and blood; generally there is painful purging, sometimes of bloody matters; swallowing is often very painful; there is excessive thirst; the abdomen becomes tumid; the pulse is small and very frequent; the urine is usually suppressed; finally, the vital powers give way, and death takes place in a period ranging from a few hours to a few days. The above symptoms will be recognized as those which (with a few exceptions) usually attend a severe case of gastro-enteritis. In truth, this is the disorder produced by this division of the irritant poisons. The action of the *corrosives*, in their undiluted state, is, of course, much more violent than that of the ordinary irritants, since they occasion immediate destruction and disorganization of the tissues, by virtue of their chemical affinities. Thus, after swallowing one of the strong mineral acids or alkalies, we find among the matters vomited, shreds of mucous membrane, detached from the œsophagus, through the corrosive action of the poison.

Where the dose of the irritant or corrosive is small, and the poison diluted, the effects are naturally much less violent. Partial or complete recovery may occur; but very frequently, especially in the case of a corrosive, the patient dies after months or years of suffering, from stricture of the gullet,—the result of the morbid action of the poison on the lining mucous membrane of this organ.

The post-mortem lesions occasioned by these poisons are generally confined to the gastro-intestinal mucous membrane. In the case of the *corrosives*, the mucous lining of the lips, cheeks, and throat exhibits patches of different colors, occasioned by the contact of the powerful agent; this membrane is softened, and reduced to a pulpy condition, and in places is entirely detached. In the stomach, there may be one or more perforations, through which the contents have escaped into the peritoneal cavity; around these openings the tissue will probably be softened and corroded. The lining membrane of the intestines exhibits, though in a less degree, the marks of the same violent action. The blood is dark-colored and fluid.

SECTION I.

POISONING BY SULPHURIC ACID.

In the five years 1852 to 1856, seventy-seven cases of poisoning by the mineral acids were registered in Great Britain: of these, seventy-three were by sulphuric acid, two by nitric acid, and two by hydrochloric acid. (Guy's Forensic Medicine, p. 394.)

SULPHURIC ACID, or *Oil of Vitriol*, in the concentrated state, is a heavy, oily-looking liquid, usually of a light-brownish color; sp. gr. 1.845; of a strong acid taste and powerful acid reaction; it speedily chars organic substances. When diluted with water, it loses its oily character, and its power to destroy organic substances. When mixed with one-half its weight of water, it occasions very considerable elevation of temperature.

Although instances of poisoning by this acid are far more frequent than by either nitric or muriatic acid, it is comparatively rarely administered with criminal intent. It is far more frequently taken by suicides, or accidentally. Children have been destroyed by its being poured down their throats; and several cases are reported where persons in a state of intoxication were murdered in a similar manner. At least two cases have been reported in which it was administered by the rectum, through mistake; also one case in which it was injected into the vagina intentionally, with a view of procuring abortion.

In the case of infants, the act is generally homicidal, and possibly accidental; in young children, accidental; in adults, nearly always suicidal. Sulphuric acid is also employed by malicious persons, by throwing it upon others, for the purpose of disfigurement of their person or destruction of their dress.

Symptoms.—When swallowed in the concentrated state of oil of vitriol, the symptoms come on *immediately*. In the act of swallowing, the person experiences a severe burning pain in the throat and gullet, and reaching to the stomach, producing the greatest agony. There is an escape of gaseous and frothy matter from the mouth, followed by retching and vomiting of matters that are powerfully acid, mixed with shreds of mucous membrane, and altered blood of a dark-brown or black color. The lips and inside of the mouth are highly corroded, presenting a whitish appearance, resembling soaked parchment. Around the lips and on the neck may be found spots of a brown color, due to the acid. There is great difficulty in speaking and swallowing; the mouth is filled with viscid mucus. The pain in the abdomen is excruciating; the stomach excessively irritable, rejecting everything swallowed. As the case advances, the respiration becomes embarrassed; the skin cold and clammy; the countenance haggard; the pulse rapid and feeble. The bowels are usually constipated, and the urine scanty. The intellect generally remains unimpaired to the last. Death occurs in a period varying from a few hours to a few days or weeks.

The above are the usual symptoms attending an ordinary case of poisoning by sulphuric acid; but there are several important exceptions to be noticed. Thus, when the acid has been poured from a vial or a spoon into the back part of the throat of a child while lying on its back, as in a case mentioned by Dr. Taylor, the mouth may entirely escape the chemical action of the poison. In other cases, its force appears to be spent upon the upper part of the larynx, causing rapid and fatal asphyxia, and none of the poison getting into the stomach. Again, cases are reported in which the vomiting was delayed for three-quarters of an hour, and was then excited only by the liquids administered. The quantity of the concentrated acid swallowed was two ounces. (Ed. Month. Jour., 1850, p. 538.)

If the acid be taken in the diluted state, its effects, although the same in kind, are less in degree, and they are less prompt in appearing. The degree of dilution may be so great as entirely to prevent its acting as a corrosive; its effects are then merely those of an ordinary irritant.

The matters first vomited contain the greater part of the poison, and are highly acid. If they fall upon marble, as on a hearthstone, they will occasion effervescence, from the escape of carbonic acid from the stone. Should they fall upon articles of clothing, they produce spots or stains, which may subsequently become valuable evidence of the administration of the poison. The color of these stains depends on the color of the stuff: thus, on black cloth they are at first red, and afterwards brownish red, and they retain their moisture for a long time. On other colored substances they produce a bright-red color; and on some others, again, a yellowish stain.

The question whether a person, after having swallowed a fatal dose of this acid, can exert any voluntary powers of locomotion, has been settled in the affirmative in several cases. These are reported as having been able to walk a considerable distance, and one as even going up the stairs of a hospital. Death frequently comes on suddenly, after a seeming remission of the severe symptoms.

Fatal period.—In fatal cases, death commonly takes place in from twelve to twenty-four hours. As just mentioned, it may occur suddenly and unexpectedly when the patient has been supposed to be recovering. In cases of perforation of the stomach, it is more rapid—in four hours; and where its action is spent upon the *rima glottidis* at the opening of the larynx, the fatal result may be almost immediate, as in the case of the child mentioned by Sir R. Christison (On Poisons, p. 132), where it was ascertained that none of the poison had entered the stomach. Other cases are reported in which death occurred in one, two, and three hours. On the other hand, the fatal result, even in acute cases, may be protracted for several days. This difference in duration may doubtless be ascribed to the full or empty condition of the stomach at the time of swallowing the acid, and also to the fact of the immediate rejection, by vomiting, of the greater part of the poison, or the contrary.

In chronic cases, when the individual has escaped the immediate fatal consequences, death may not result till after the lapse of several months; and it would seem to be immediately owing to inanition, from stricture of the œsophagus, or from chronic inflammation of the stomach. The most protracted case on record is the familiar one mentioned by

Dr. Beck (Medical Jurisprudence, ii. p. 472), in which the patient died *two years* after taking the acid, from stricture of the œsophagus.

Fatal quantity.—The smallest fatal dose for an adult recorded is one drachm,—a case quoted by Sir R. Christison,—where a stout young man died in seven days after swallowing this amount. The same authority mentions another case, of an infant, which was destroyed by swallowing half a drachm of the concentrated acid. The danger appears to depend more on the degree of concentration of the poison than on its absolute quantity. Several cases are recorded of recovery after swallowing as much as one, two, and, in one instance mentioned by Dr. Beck (*loc. cit.*, vol. ii. p. 468), *four ounces* of the strong acid.

Treatment.—On account of the immediate corrosive action of this acid, chemical antidotes can rarely be employed in time to prevent serious injury. These antidotes consist of solutions of the alkaline carbonates in water or milk, magnesia or chalk suspended in milk, soap-suds, the scrapings from a whitewashed wall (in the absence of the other articles), and oily emulsions. Dr. Taylor considers the solutions of the alkaline carbonates preferable to either chalk or magnesia, in consequence of the insoluble particles of the latter adhering closely to the coats of the stomach, and thus not coming into immediate contact with the acid. Sometimes it is almost impossible to make the patient swallow, in consequence of the disorganized condition of his throat: in such a case the very cautious use of the stomach-pump may be advisable; but care should be taken to avoid perforation of the œsophagus. In cases of threatened suffocation, tracheotomy should be performed. In all cases it will be necessary to combat the violent inflammatory symptoms with the usual appropriate remedies. Among the symptoms mentioned as having occurred, particularly in some of the non-fatal cases, is profuse salivation; and, in some instances, masses of false membrane, moulded into the form and size of the œsophagus, have been expelled by coughing.

Post-mortem appearances.—In poisoning by sulphuric acid, it may happen, as already observed, that the lining membrane of the mouth entirely escapes the corrosive action, owing to the manner in which the poison was administered, viz., pouring it from a spoon far back into the throat; so, on the other hand, fatal cases have occurred in which the corrosive action has been entirely confined to the mouth and throat, and the stomach has completely escaped. In all cases a thorough examination of the whole alimentary tract should be made, from the mouth downwards. The mucous membrane of the mouth and throat, including the tongue, will be often found whitish, thickened, and softened. The lining of the œsophagus may be greatly corroded, detached in folds, and of an ashy-gray color. The *stomach*, when not perforated, is collapsed and contracted. Its contents are often of a dark-brown color and tarry consistence, composed of mucus and altered blood. They may, or may not, be acid, depending on the time elapsed and the antidotes administered. The mucous membrane of the stomach exhibits all the evidences of intense inflammation, such as striæ of deep redness, softening, and thickening. Portions of it may be also detached. When perforation has occurred, the coats are softened, and the edges of the aperture are apt to be black and irregular. If the contents have escaped into the abdomen, the surrounding parts are blackened and corroded by the poison. It is probable that, in some instances, the perforation of the stomach takes place after death, from the chemical action of the acid.

If the case is much protracted, the *small intestines* may exhibit very decided marks of corrosion, and even of perforation. It should be remembered that after death from *dilute* sulphuric acid, although no marks of corrosion may be discovered, the evidences of inflammation will be sufficiently distinct. An instructive case is quoted by Dr. Taylor from Journ. de Chimie Méd., 1846, ii. 17, where an infant aged two months died from the effects of dilute sulphuric acid. There was an entire absence of all marks of corrosion on the lips, tongue, and throat, œsophagus, and stomach; and no very great amount of inflammation was observed in the stomach. Moreover, no trace of the acid could be discovered in this organ by chemical analysis; but it was proved to exist abundantly in spots on the clothing. The prisoner was convicted, and sentenced to hard labor for life.

According to Casper (Foren. Med., vol. ii. p. 58), the bodies of persons poisoned by sulphuric acid resist decomposition for a long time. This effect is attributed by him to the neutralizing of the ammonia resulting from the putrefaction, by the acid.

Absorption and elimination.—As regards the question whether this acid is absorbed into the circulation and eliminated by the emunctories, the cases and experiments reported go to prove the affirmative. Casper states that in cases examined by him, the blood, in every instance, had a cherry-red color, was of a ropy consistence, and had an acid reaction. A case is mentioned by Dr. Beck (Med. Jurisp., ii. p. 475) of a pregnant woman dying from the effects of sulphuric acid, in whom the amniotic fluid, as well as that of the pleura, peritoneum, heart, and bladder of the fœtus, had an acid reaction. It has also been detected in the urine, during life.

Chemical analysis.—The concentrated acid (oil of vitriol) possesses the following properties: 1. It immediately chars and blackens organic bodies, such as wood, cork, sugar, etc. 2. Boiled with wood chips, copper, or mercury, it gives off sulphurous acid fumes, easily recognized by their odor, and by their bleaching effect on colors. 3. When mixed with its own volume of water, it gives out great heat, raising the temperature nearly to 212° F.

The diluted acid is readily detected by a soluble salt of barium—either the chloride or the nitrate. To the suspected liquid a few drops of nitric acid are first added, and then a little of the solution of chloride of barium; an immediate copious white precipitate subsides—the *sulphate of baryta:* this is insoluble in all acids and alkalies. But the mere obtaining of such a white precipitate is not *positive* evidence of the presence of sulphuric acid, since selenic and hydrofluosilicic acids produce similar results with a barytic salt. In order to confirm this test, the precipitate should be dried, and thoroughly mixed, either with twice its weight of powdered charcoal, or with equal parts of carbonate of soda and cyanide of potassium, or with four or five times its weight of well-dried ferrocyanide of potassium, and heated to redness on platinum-foil, or in a reduction-tube. This converts the sulphate into a sulphuret: when cooled, the residue is moistened with dilute hydrochloric acid, when the odor of sulphuretted hydrogen is immediately recognized,—proving the presence of sulphur in the original acid. Or, the residue may be put upon a moistened glazed card (containing carbonate of lead), when it will produce a brown or black stain (sulphide of lead). Or, the residue may be put into a watch-glass and moistened with the dilute acid, and covered over with another glass containing a fragment of paper moistened with a solution of acetate of lead: the latter will very speedily assume a dark-brown color. By any of the above means this acid can be positively recognized, even in very small proportions. A solution containing only the one-twenty-five-thousandth part of its weight of sulphuric acid is precipitated; and less than the one-hundredth part of a grain of the acid will yield sufficient precipitate to admit of the application of the confirming test. (Wormley.)

If the precipitate in the above cases had been due to selenic or hydrofluosilicic acid, on ignition there would, of course, be no production of a sulphuret; which would prove the absence of sulphuric acid.

Two circumstances require attention in the application of the baryta test: (1) this test yields a similar result when applied to a solution of any *sulphate;* (2) it throws down also a white precipitate with several other acids when in combination, *e.g. carbonic*, *phosphoric*, *boric*, *oxalic*, etc.

In regard to the first, while it is true that a solution of alum, of Epsom salt, or of any other sulphate will precipitate chloride of barium, the presence of any saline matter may easily be detected by slowly evaporating a drop or two of the original solution on a watch-glass, when a residue will be left from a saline solution; whereas in a purely acid solution there will be none. But a case may present where, along with free sulphuric acid, there is present some medicinal sulphate, such as Epsom or Glauber's salt. Here, the simple baryta test would lead to an erroneous inference as to the actual amount of *free* sulphuric acid present. The error may be obviated by precipitating all the free acid by means of finely-powdered carbonate of baryta, first warming the liquid; until effervescence ceases (the carbonate does not decompose any sulphate that may be present). The precipitated sulphate of baryta will represent the *free* sulphuric acid only. Again, it might happen that in a suspected acid solution there is present some sulphate, as Epsom salt, and some other acid, as citric, tartaric, etc.: here, of course, baryta would give the appropriate reaction; but the fact of the absence of *free* sulphuric acid can be shown here by first evaporating a portion of the liquid to dryness: *a residue* will indicate some saline substance. The further mode of proceeding will be explained *post* (p. 145).

(2) As regards the precipitate occasioned by baryta in salts containing phosphoric, carbonic, oxalic, etc., acids, a simple test will serve immediately to distinguish between these and a sulphate of baryta: the addition of either hydrochloric or nitric acid will instantly redissolve the former, while no effect is produced on the latter.

Another delicate test for the presence of dilute sulphuric acid is *veratria.* This alkaloid, according to Prof. Wormley (Micro-Chem. of Poisons, p. 112), is much more delicate than the cane-sugar test of Runge: it will detect with certainty so small a quantity as the one-thousandth of a grain. A small portion of the alkaloid is introduced into the diluted acid, and carefully evaporated to dryness; a beautiful *crimson* deposit is left. Moreover, as this test produces no effect with neutral sulphates, but only with the *free* acid, it serves to distinguish the latter from the acid in combination.

Detection in organic matters.—These, if thick and turbid, should be boiled with distilled water, and filtered through paper supported by muslin. Mere color is no obstacle, provided the solution is clear. A measured quantity of the liquid, concentrated, if necessary, should then be acidified with nitric acid, and treated with a solution of chloride of barium, until it ceases to precipitate. The mixture is then warmed and filtered; the deposit washed in the filter with water containing hydrochloric acid, and dried. In medico-legal cases it will always be proper to confirm this reaction by reducing the suspected sulphates, in the manner pointed out on page 143. Other parts of the solution may be submitted to the confirmatory tests.

Although by this process the presence of sulphuric acid can be certainly established, yet it does not follow that this acid existed in the *free* state, even when the liquid had a strong acid reaction; for it might happen that some neutral sulphate was present along with any common acid, like acetic, citric, etc.—vinegar, for instance—or some acid sulphate, as alum; or, it might be, a mixture of free sulphuric acid and a sulphate. Now, in each of the above supposed cases the baryta test would give precisely similar results: consequently the baryta test alone cannot be relied on for detecting *free* sulphuric acid, in a medico-legal case.

If, on evaporating a portion of the clear liquid to dryness, no residue is left, it is certain that the acid existed in the free state; if, however, a saline residue is left, then there is no positive evidence of the presence of any free acid. Moreover, when there has been much organic matter in the original liquid, it may be difficult to determine whether the residue is saline or organic. In this case, the suspected organic matter must be destroyed by repeatedly moistening the residue with pure strong nitric acid and evaporating to dryness by a moderate heat until the residue has a yellow color, after which the heat is raised until the organic matter is entirely destroyed, when, if a salt be present, it will remain as a white mass. If this residue, on examination, proves to be a sulphate, we are still unable to say whether *any* sulphuric acid was present in the free state.

In a case like the above, where we wish to determine whether the whole of the acid existed as a sulphate, the following process is recommended: A given volume of the solution is acidulated with hydrochloric or nitric acid and precipitated by an excess of chloride of barium, the precipitate washed, dried, and weighed. An equal volume of the original solution is evaporated to complete dryness, in order to dissipate any free sulphuric acid, and then dissolved in acidulated water, filtered, and precipitated as before; and the dried deposit weighed. If no free sulphuric acid was present, the weight of each of the barytic precipitates would, of course, be equal; whereas, in the other case, the weight of the former precipitate ought to exceed that of the latter, by exactly the amount of the free acid.

MM. Tardieu and Roussin (Sur l'Empois., p. 194), after reviewing the various methods proposed for determining free sulphuric acid when associated with a sulphate, recommend the following process. The object is to saturate the free acid with a base, the sulphate of which is soluble in alcohol; this base is *quinia* recently prepared. A clear solution of the acid sulphate of quinia is first precipitated by ammonia in slight excess; the hydrate of quinia is washed with distilled water until the washings yield no precipitate with chloride of barium. In the mean time, the suspected organs, vomited matters, etc., which have been digested in warm distilled water for a sufficiently long time, are filtered, and the filtrate put into a porcelain capsule. The quinia is now added in slight excess, and the whole evaporated on a water-bath. The semi-liquid extract which remains is treated several times with absolute alcohol, which dissolves out the *sulphate of quinia* formed at the expense of the free acid, and leaves the other matters (sulphates) untouched. The alcoholic solutions are filtered, and evaporated anew; and the resulting extract is dissolved in a small quantity of boiling distilled water, and immediately filtered. If the amount of the free sulphuric acid is at all considerable, the sulphate crystallizes on cooling, but if the quantity be too minute to form crystals, it will still be easy to prove the presence of sulphuric acid by the baryta test.

It may happen that, in consequence of the alkaline antidotes administered, the matters vomited, as also the contents of the stomach, have a neutral reaction, the acid existing only in the form of a sulphate. In such a case, after proceeding in the usual way, and obtaining a clear solution, the precipitate obtained by chloride of barium would, of course, indicate only the quantity of the *combined* acid; and the chemical analysis *alone* could not furnish proof of poisoning. Certainly, if the quantity of combined acid thus discovered was small, no inference whatever could be drawn from the experiment, since the small amount of sulphates normally present in the tissues of the stomach and in the food, as well as any sulphate accidentally present, would satisfactorily account for a small deposit of sulphate of baryta.

M. Tardieu recommends, in such a contingency, to ascertain, as well as possible, the nature of the substances given to counteract the poison, as well as of the food last taken by the deceased. With a knowledge of these facts in his possession, the expert prepares a mixture of meats and articles resembling those supposed to have been present in the stomach; of these, he uses a quantity equal in weight to that of the vomited matters, or of the stomach and its contents: these two masses are dried, and then put into two separate crucibles of the same size, which are heated to redness, until empyreumatic fumes cease to be given off, and the organic substances are charred. These masses of carbon are then to be separately powdered, and each mixed with a fourth of its weight of pure powdered nitrate of potash, free from any sulphate. Each of the masses is then projected, in small quantities, into a red-hot crucible, and, after deflagration has ceased, the cooled residues are dissolved in distilled water. The solutions are strongly acidulated with nitric acid, and treated with an excess of chloride of barium. The two precipitates are washed, filtered, dried, burned, and weighed. If their weight is about equal, the conclusion would be against the idea of poisoning; but if, on the contrary, that of the precipitate derived from the stomach and its contents is decidedly greater than that of the other, then the presumption is very strong in favor of poisoning (*loc. cit.* p. 196).

From the above considerations it is evident that it will not always be in the power of the toxicologist to prove a case of poisoning by sulphuric acid merely by the chemical analysis: he must seek further evidence in the well-marked symptoms, and the post-mortem signs, together with the circumstances of the case.

Detection of spots on clothing, etc.—The stains produced by sulphuric acid on articles of clothing, etc., are easily recognized. The texture of the fabric with which the acid comes in contact is more or less destroyed, and the color more or less changed: on black cloth the stain exhibits a red-brown tint; some colors it changes to yellow. The strong acid chars and blackens white linen and cotton fabrics. Although the dilute acid does not blacken these articles, yet when impregnated with it they become charred on exposure to a moderate heat. According to Dr. Taylor, the color of black leather is not changed by sulphuric acid. Likewise, articles dyed with either indigo or Prussian blue do not change their color by it (nitric acid changes the color of indigo to yellow). Another fact in connection with these stains is, that they remain moist for a long time, in consequence of the affinity of sulphuric acid for water.

In order to examine the spots chemically, one or two of them should be cut out and soaked for awhile in hot distilled water. A brown-colored solution is obtained, which has an acid reaction, and responds to the usual baryta test. It should, however, be remarked that an acid sulphate will cause a similar stain upon cloth. The salt may be detected in the cloth by incineration. In all such examinations of suspected stains, a portion of the unstained fabric should likewise be examined for the presence of the acid, inasmuch as many articles of clothing contain slight traces of sulphates, though not of free acid.

Quantitative analysis.—Sulphuric acid is usually estimated as sulphate of baryta. The precipitated sulphate, after repeated washing with hot water acidulated with hydrochloric acid, on a filter, is dried, ignited, and weighed, allowance being made for the ash of the filter. One hundred parts of the sulphate are equal to 42.02 parts of monohydrated sulphuric acid.

The following results of different cases reported in the English and foreign journals are taken from Guy's Forensic Medicine, p. 402:

Of 36 cases (the majority females)—26 were fatal (all the children, and 18 adults); and 10 recovered (all adults).

Of 31 cases—20 were suicidal; 3 homicidal (all young children); and 8 accidental (2 of them children).

Among adults, both in accidental and suicidal poisoning, there was 1 recovery to 2 deaths.

Of the 26 fatal cases, 10 lasted a day or less; 6 more than a day, and less than a week; 3 less than two weeks; 1 from two to three weeks; 1 over three weeks; and 5 from five to forty-five weeks.

The least duration in 5 children was three and a half hours; the greatest, three days. In 20 adults, the least was three and a half hours; the greatest, forty-five weeks.

The recoveries took place in from 6 to 20 days.

Perforation of the stomach occurred in 8 cases out of 21, in which the post-mortem appearances are described.

SECTION II.

POISONING BY NITRIC ACID.

Nitric acid (*Aqua fortis*) as found in commerce is a powerful corrosive liquid, having a yellow or orange color, and a density varying from 1.35 to 1.45. It is apt to be contaminated with sulphuric acid, chlorine, and iron. In its chemically pure state, it is colorless. Although very much employed in the arts, it is very rarely the occasion of poisoning. The cases that do occur are usually the result of accident, or of suicide. Orfila relates a case in which a man poured a spoonful of the acid into the ear of a drunken wife, while asleep, which was ultimately the cause of her death, after the lapse of about seven weeks. This, however, cannot be regarded properly as a case of poisoning.

Symptoms.—These are almost identical with those produced by sulphuric acid. There is the same immediate burning pain on swallowing; the violent pain in the abdomen—perhaps more diffused than that produced by sulphuric acid; the gaseous eructations also more copious; similar vomiting; often violent purging, the dejections being mixed with blood. Sometimes there is obstinate constipation, with suppression of urine. The difficulty of respiration is often extreme, rendering the operation of tracheotomy necessary. The lips and inside of the mouth, and the tongue, present at first a whitish appearance, which soon becomes of a yellowish hue, and ultimately brown. When drops of the acid have fallen upon the cheeks, neck, or other parts of the body, a permanent yellow stain is produced. The teeth are white, but yellowish at their junction with the gums. The subsequent symptoms are those of collapse, such as a small, frequent, and feeble pulse, cold skin, and great prostration, and sometimes a sort of stupor. The intellect is usually unaffected. As in the case of sulphuric acid, the poison may spend its violence on the respiratory organs, and never get into the stomach.

The points that distinguish the symptoms of this poison from those of sulphuric acid are the yellowish color of the stains, both in the mouth and on the body; the rather less violent local action of nitric acid on the mucous membrane, and the occasional purging of bloody matters.

Nitric acid gives off powerfully irritating fumes at ordinary temperatures, which occasionally produce fatal effects when breathed. In these instances, after death, the mucous lining of the trachea and bronchi were found deeply congested. Death was probably occasioned by œdema of the glottis. The vapors from a mixture of nitric and sulphuric acids—a compound much used in the arts—are extremely dangerous to breathe. In these cases, the violent symptoms may not come on for several hours, but, after appearing, they progress with extreme rapidity, and terminate fatally in the course of ten to fifteen hours.

Fatal dose.—In the fatal cases of poisoning by this acid, the quantity taken has not been mentioned. Authorities, however, unite in saying that two drachms of the concentrated acid would prove fatal. A smaller dose would destroy life in infants. Yet much larger doses have been taken with ultimate recovery.

Fatal period.—Dr. Taylor records a case where death occurred in one hour and three-quarters after swallowing the poison; while, on the other hand, Tartra relates an instance which did not prove fatal for eight months. The usual fatal period is within twenty-four hours. Out of fifty-six cases of poisoning by nitric acid collected by Tartra, twenty-one completely recovered, and eight partially—making the mortality about one-half.

Treatment.—This is essentially the same as that recommended for sulphuric acid (*ante*, p. 140).

Post-mortem appearances.—In acute poisoning by nitric acid, the inside of the lips, mouth, and throat, together with the tongue, will usually present a yellow hue; sometimes it is grayish white, and more frequently brownish; sometimes large patches of the membrane are destroyed. The lining membrane of the œsophagus is also of a yellowish color; it is generally thickened and softened, and sometimes removed in longitudinal streaks. A similar appearance will be noticed about the glottis and the interior of the larynx and trachea, if the acid has gained access to these organs. As in the case of sulphuric acid, it may happen that none of the poison has passed into the stomach, but the whole force has been spent upon the respiratory organs. The examiner should not fail to notice particularly any spots or stains upon the face, neck, and other exposed parts of the body, as well as upon the dress. These spots, if due to nitric acid, will exhibit a decided yellow color. Those on the clothes, after a time, become brownish, and, unlike the spots made by sulphuric acid, they become dry, rotting the texture. Moreover, as the acid is volatile, if the examination of the stains be deferred for any length of time, it will be impossible to identify them. A few weeks suffice to remove all chemical traces of this acid, differing in this respect vastly from sulphuric acid, the stains of which, on articles of clothing, remain unaltered and distinguishable for many years.

The stomach is distended, often exhibiting on its outer surfaces patches of green color, due to the action of the acid on the bile. Too much stress must not be laid upon this appearance, since in certain diseased states of the body the bile itself undergoes a change which imparts to it a very similar green color. The stomach is sometimes adherent to the other viscera, and cases are reported in which this organ was completely destroyed by the corrosive action of the acid, a cavity being left among the surrounding viscera, which contained coagulated blood. The contents are usually colored yellow. The mucous membrane is softened, thickened, detached in patches, deeply congested,—the vessels being injected with dark coagulated blood. Perforation rarely occurs, but when it does, the contents of the stomach pass into the peritoneal cavity, imparting a yellowish tint to the adjacent viscera, and also producing intense peritoneal inflammation.

The upper portion of the small intestines will be very likely to exhibit appearances similar to those seen in the stomach; but these may at times altogether escape corrosion.

The large intestines are not apt to be affected; and, unless in the exceptional cases where purging has existed before death, they may contain hardened fæces. The other abdominal organs are usually inflamed, even when the stomach has not been perforated. The lungs and bronchi are deeply congested in those cases where the force of the poison has been spent upon the larynx and trachea.

In cases of death from *chronic* poisoning, the body is greatly emaciated, the stomach and bowels more or less contracted and thickened; in one instance reported, where the patient lived three months, the pylorus and the upper part of the duodenum were contracted to the diameter of one or two lines; the mucous membrane was softened and red in patches, and there were several cicatrices of ulcers. (Med.-Chir. Rev., vol. xxviii. p. 553.)

Chemical analysis.—The concentrated acid may be recognized, (1) by its giving out white fumes when exposed to the air. (2) By its staining organic substances yellow or brown, —the color being heightened by touching with a drop of caustic alkaline solution. (3) By its powerful action on metallic copper, evolving copious orange-colored pungent fumes, and leaving a blue solution (nitrate of copper). Other metals are acted on with equal energy, as mercury, zinc, tin, etc. This action of nitric acid, in the cold, upon metals, with the evolution of the red fumes, is quite characteristic. If the acid be diluted, it may require to be boiled before its action on copper will be visible. (4) It does not dissolve gold-leaf, even on boiling; but if hydrochloric acid be added, the gold is immediately dissolved.

The dilute acid.—On account of the free solubility of the nitrates, no precipitate can be obtained by the use of reagents, as in the case of the other two mineral acids; nevertheless, there is no difficulty in identifying it. (1) Unless very much diluted, when boiled on copper clippings, it yields the characteristic red fumes, and leaves behind a blue solution. (2) When neutralized by carbonate of potash, and a piece of filtering-paper is dipped into the resulting solution (nitrate of potassa) and dried, it burns like touch-paper when applied to a flame. (3) If this solution be evaporated, it will yield characteristic lengthened striated prisms of *nitrate of potassa.* If neutralized with soda, the crystals present the well-known rhombic form of the *nitrate of soda.* A single drop of the solution evaporated on a glass slide will exhibit, under the microscope, the well-marked characters of each of these salts. (4) A fragment of these crystals is put into a very small test-tube along with a grain or two of copper filings, then moistened with water, and a few drops of sulphuric acid added: either with or without heating, the evolution of the orange vapors and the production of a blue liquid will prove the presence of nitric acid. (5) Proceed as in 4, substituting for copper a small crystal of *morphia:* it will yield an orange color and a yellowish liquid; the color becomes fainter by boiling. (6) As in 4, substituting for copper a fragment of *brucia:* a blood-red color is the result, which is removed by the application of chloride of tin. (7) As in 4, using a crystal of *narcotina* in place of copper: a reddish-brown color is produced, changing by gentle heat to a blood-red. (8) Mr. Horsley has proposed a test, the delicacy of which is confirmed by Prof. Wormley,—*pyrogallic acid;* it is employed as follows: a small quantity of water acidulated with a few drops of sulphuric acid is put into a small test-tube; add a fragment of pyrogallic acid; after which a little concentrated sulphuric acid is allowed to flow down the inside of the tube so as to subside to the bottom; add a few crystals of common salt, and, when effervescence ceases, drop in a fragment of the suspected nitrate; the acid at the bottom of the tube very soon assumes an intense purple hue, which may extend to the rest of the liquid. (9) *The iron test.*

—Add to a few drops of the dilute acid, or to a fragment of a nitrate, in a small test-tube, a large excess of pure concentrated sulphuric acid; heat the mixture for a short time, and then cool it by immersing the tube in cold water (it is essential that the mixture should be cold); then allow a few drops of the fresh solution of protosulphate of iron to flow down the inside of the tube; at the line of junction of the two liquids there will be formed a beautiful purple, or brownish-purple, zone,—the intensity of the color depending on the quantity of nitric acid present. This color will extend throughout the liquid, on gently stirring it, so as not to evolve heat. On subsequently heating the tube, the color disappears, with the evolution of the characteristic red fumes. This is a very delicate and satisfactory test, if properly made. Care should always be taken to insure the purity of the sulphuric acid, as even the purest samples are apt to contain traces of nitric or nitrous acid; in which case it would give the above reaction with the iron solution without the addition of any nitric acid. (10) *The indigo test.*—When a weak solution of sulphate of indigo (indigo dissolved in sulphuric acid) is heated in contact with nitric acid (or a nitrate and sulphuric acid), the color disappears. (11) *The gold test.*—Add to the suspected solution, concentrated, or to a fragment of the crystal, a few drops of strong hydrochloric acid along with a slip of gold-leaf, and apply heat; the gold will be dissolved as a chloride. The presence of gold may be recognized, if not in too minute quantity, by protochloride of tin, which produces a purple precipitate, or at least imparts a purple color to the liquid. As hydrochloric acid frequently contains chlorine, it should first be tested alone with gold-leaf, before adding it to the suspected nitrate.

A similar reaction is also given by chlorates, hypochlorites, chromates, iodates, and bromates; also by the sesquisalts of iron (H. Wurtz, Chem. Gaz., xvii. p. 32).

Several other tests have been proposed; but, as they are of inferior value to those detailed above, they need no further notice.

Detection in organic mixtures, contents of stomach, etc.—If the mixture is viscid, or contains solid matters, it should be gently boiled in distilled water for about twenty minutes, allowed to cool, and then filtered; the matters on the filter thoroughly washed; and the liquid concentrated by evaporation. If found to be acid on testing it with litmus-paper, it should be neutralized with bicarbonate of potassa, and allowed to crystallize by evaporation. If the resulting crystals are much discolored by the organic matters, the latter may be removed to a great extent by absolute alcohol, which has no material effect on the crystals. These may now be dissolved in a very small quantity of pure water, and re-crystallized by evaporation. A few drops of the solution allowed to dry upon a glass slide and examined with the microscope will exhibit the characteristic six-sided striated crystals of nitrate of potassa. The different tests already mentioned may then be successively applied.

If the original solution contains enough of the acid, the copper and gold tests may be applied directly.

It may happen, however, that in consequence of the antidotes administered—lime or magnesia, or one of the alkaline carbonates—the matters examined are no longer acid, but neutral; the free acid has combined with a base, and now exists as a nitrate. Under these circumstances, the solution, after boiling, if necessary, and filtration, should be treated with bicarbonate of potassa (or soda), and heated for a short time, to allow the insoluble carbonate of lime or magnesia to subside. It is then filtered, and the filtrate containing the nitrate of potassa is allowed to crystallize by evaporation, and the crystals examined, as before mentioned. If the antidote administered was either carbonate of potassa or soda, it will only be necessary to evaporate the clear filtered liquid in order to procure crystals of the nitrates of these alkaloids, which can be tested as above.

It should be remembered that although the contents of the stomach, or rather organic matters, may have an acid reaction, and exhibit the evidences of the presence of nitric acid, yet this acid may not exist in the free state, as *e.g.* when some nitrate happens to be present along with an ordinary acid. In such a case, the method recommended for sulphuric acid (*ante*, p. 145) is to be pursued. If the acid exists in the free state *only*, on evaporating a few drops of the original filtered solution, no saline residue should be left.

Orfila considers the *sulphate of narcotina* and the *protosulphate of iron* (iron test) as two of the most delicate tests for nitric acid. His mode of employing them is as follows. A minute fragment of the suspected nitrate is mixed with copper filings (or a small piece of copper-foil) and put into a very small test-tube with a drop or two of water and five or six drops of pure concentrated sulphuric acid: a small glass tube bent at right angles attached to the test-tube serves to conduct the disengaged gas into another small test-tube containing a few drops of a solution of sulphate of narcotina. On heating the first tube, the deutoxide of nitrogen will pass over into the second one, and immediately produce a blood-red color. If the second test-tube contains a solution of the protosulphate of iron, the disengaged gas will immediately cause it to assume a dark-brown or coffee color; if now five or six times its volume of strong sulphuric acid be added, it will acquire a violet or purple hue. (Toxicologie, 1852, i. p. 178.)

Although these two tests are so very delicate, yet they cannot be regarded as *always* indicating the presence of nitric acid, to the exclusion of all other bodies. Orfila himself tells us (*loc. cit.*, p. 181) that healthy urine will redden narcotina, and produce a brownish color with the iron, which subsequently turns violet on the addition of sulphuric acid. Urea acts in the same manner; and the serum of the blood drawn from a patient affected with pleurisy, although not acting on the narcotina, produced upon the iron-salt precisely the same effect as that caused by nitric acid.

Orfila further ascertained that if the nitrate is present in very small quantity, along with a large excess of alkaline chlorides, or of organic matters, the copper and sulphuric acid test fails to produce a gas which will give the characteristic reaction with protosulphate of iron (*loc. cit.*, p. 182).

Absorption and elimination.—According to Orfila, nitric acid is absorbed into the general circulation, and eliminated by the urine. He proved its presence in this secretion, in animals poisoned by the acid, by distilling the urine with sulphuric acid, and neutralizing the distillate with potassa: on evaporation, the characteristic crystals of nitrate of potash were obtained. This result does not uniformly follow, but occurs only during certain stages of the poisoning.

In examining a body poisoned by nitric acid after several months' interment, the poison will, in all probability, be found to have entirely disappeared, having been converted into *nitrate of ammonia.* But as this salt sometimes occurs in the soil, its presence in the body might be ascribed to percolation from the ground in which it was buried. In such a case, a portion of the surrounding soil should be analyzed. If putrefaction be not too far advanced, it may be possible to recognize some of the marked post-mortem changes in the alimentary canal.

Examination of suspected stains.—These should be cut out, and soaked for a time in warm distilled water. The presence of a free acid having been tested by litmus-paper, the liquid should be neutralized with bicarbonate of potassa, and filtered, and the resulting crystals examined by the microscope and subjected to the appropriate tests (see *ante*, p. 153): the dried filter burns like touch-paper. As nitric acid is volatile, it is soon dissipated, by exposure, from the substances on which it has fallen. After a certain length of time, the spots on woolen or cotton garments turn brownish, become dry and rotten, and lose every trace of the acid. Sir R. Christison was able to detect its presence in stains seven weeks old; and Dr. Guy quotes an instance in which it was discovered, under similar circumstances, after an interval of some months (Wormley).

To distinguish stains of nitric acid from those caused by iodine or bile, test with a weak solution of caustic potassa: the nitric acid stain assumes a bright orange tint, while the iodine (or bromine) stain immediately disappears; and the bile stain undergoes no change.

SECTION III.

HYDROCHLORIC ACID (MURIATIC ACID).

This acid, sometimes known in commerce by the name of *spirit of salt*, occurs usually as a light-yellow liquid, powerfully acid, fuming when exposed to the air. It emits dense white vapors when brought in contact with the vapor of ammonia. Its sp. gr. is usually about 1.15. When chemically pure, it is colorless; its ordinary yellow color is due to chloride of iron, or chlorine, or both. The commercial acid, also, frequently contains arsenic, nitric acid, common salt, and occasionally antimony.

Instances of poisoning by this acid are comparatively rare; especially those of a homicidal character.

Symptoms.—These are, in the main, similar to those already described as resulting from sulphuric and nitric acids. Among these symptoms, Tardieu mentions the grayish appearance of the stains around the mouth and in the interior of the buccal cavity, together with the formation of false membrane upon the mucous surfaces attacked by the acid, as characteristic of hydrochloric acid (*loc. cit.*, p. 235).

Fatal dose. — The smallest quantity recorded as having proved fatal is *half an ounce.* This was swallowed by a woman aged sixty-three years, with suicidal intent, and caused death in eighteen hours (Wharton and Stillé, Med. Jurisp., vol. ii. p. 322). As in the case of the other mineral acids, recoveries have occurred after much larger doses have been swallowed, after the use of proper remedies. The period at which death takes place in acute cases varies from five hours and a half (the shortest period) to *eight days;* in the latter case, two ounces of the strong acid had been swallowed. In more chronic cases, life was prolonged for *eight weeks.*

The *treatment* is similar to that employed in the case of the other mineral acids (*ante*, p. 140).

Post-mortem appearances.—These, on the whole, closely resemble the morbid lesions produced by sulphuric acid. The throat, mouth, and gullet have been found highly inflamed, their lining membrane detached in masses, or sloughing away. The mucous lining of the stomach is extensively corroded, softened and thickened. The contents are sometimes of a yellowish, and sometimes of a greenish, color. On removing these, the lining membrane has been found blackened in ridges, as if charred, and the intervening furrows of a bright-red color. This appearance may extend through the duodenum. Perforation of the stomach is comparatively rare; though in a case reported by Dr. Galtier, this organ was entirely disorganized and softened; it presented posteriorly several perforations of different diameters, with rounded, thickened, and inflamed margins, adhering to the adjoining viscera by albuminous deposits. The pyloric orifice was thickened, as well as the mucous membrane of the small intestines. The gullet was thickened throughout, and its mucous membrane was in a state of suppuration. In this case the person survived eight weeks after taking the poison. In the more protracted cases there is a greater tendency to softening and thickening of the mucous lining of the gullet, stomach, and small intestines, with partial removal of this membrane in shreds or patches. In those cases where the force of the poison has spent itself upon the respiratory organs—the glottis, larynx, and trachea—these will be found deeply injected, often corroded, with more or less detachment of the lining membrane, together with engorgement of the lungs and pleura. In cases of this nature the stomach may possibly entirely escape the corrosive action of the poison.

Prof. Guy (Forensic Medicine, p. 408) has given some good illustrations of the morbid changes in the gullet and stomach resulting from the corrosive action of muriatic acid. The shriveled, worm-eaten appearance of the former, together with the patches denuded of epithelium, as also the black grumous condition of the inner coats of the stomach, bear a strong resemblance to the condition observed in poisoning from sulphuric acid. He also very properly cautions against mistaking for the above condition that state of the mucous membrane resulting from the effects of the acid secretions of the stomach, before and after death, more especially in chronic diseases, as phthisis; in which there may be abrasion of the epithelium, together with a similar black granular deposit, alternating with red injected streaks.

Chemical analysis.—1. *The strong acid.*—This is distinguished from the other acids, (*a*) by its yellow color; (*b*) by its giving off dense white fumes when in contact with ammonia; (*c*) by its negative action on metallic copper or mercury, either cold or boiling; (*d*) by its giving off *chlorine gas* when heated with peroxide of manganese: this last test is characteristic. It produces a greenish stain on black cloth.

2. *In the diluted state.*—The failure to cause a precipitate with chloride of barium would prove the absence of sulphuric acid. The characteristic test is *nitrate of silver* in solution: this occasions, even in the very dilute acid, a copious, curdy, white precipitate, which soon darkens on exposure to the light. This precipitate is insoluble in boiling nitric acid, and in caustic potassa, but is very soluble in ammonia. When it is dried and heated, it fuses into a yellow liquid, which, on cooling, becomes a soft, horny mass. As any soluble chloride, *e.g.* common salt, will give precisely the same reaction with nitrate of silver, a drop or two of the original acid liquid should be carefully evaporated, when, if no residue is left, the acid will have been in the free state. Nitrate of silver also produces, in neutral solutions, precipitates with other acids or elements; but all these precipitates, with the exception of that from hydrocyanic acid, are freely soluble in nitric acid. The *cyanide* of silver is easily distinguished from the chloride by fusion; it yields an inflammable gas, which burns with a rose-colored flame. (See *post*, HYDROCYANIC ACID.)

Detection in organic mixtures.—As most organic mixtures contain hydrochloric acid, free or combined, these will generally yield a whitish precipitate with nitrate of silver. In the contents of a stomach we may expect always to find more or less of this acid, as it is one of the constituents of the gastric juice. In some cases of disordered digestion, its quantity is greatly augmented. Dr. Prout once found between four and five grains of pure hydrochloric acid in sixteen ounces of the fluid of water-brash (Philos. Trans., 1824, p. 49). From this it follows that great caution must be observed as regards the inference of poisoning by hydrochloric acid, from the chemical analysis *exclusively.* Most certainly, if the symptoms and morbid lesions do not fully warrant the diagnosis, it would be very unsafe to found it upon the chemical results, for the reasons given above.

As this acid adheres with great tenacity to organic matters, it will be found almost impossible to separate it from the latter, especially if it be present in very small quantity, by the usual process of boiling, filtering, and distilling. According to Christison, it will seldom be found in the distillate. Under such circumstances, Orfila recommended to treat the residue in the retort with a strong solution of tannin, filter, and distill the filtrate, as before, to near dryness, avoiding a higher temperature than 240° F. But it must be remembered that this process will indicate hydrochloric acid in a mixture that contains merely a chloride (as common salt) and sulphuric acid. This fallacy can, however, be avoided by proving the absence of sulphuric acid by the baryta test.

Practically, the following process, recommended by Tardieu (*loc. cit.*, p. 238), is found to answer best, in medico-legal cases, where it is important to determine whether hydrochloric acid is present *in the free state* in the contents of the stomach, or in other complex organic mixtures. Any solid matters are cut into small pieces, and the mass diluted, if necessary, with distilled water, heated to near the boiling-point for about half an hour, cooled, and filtered. It is then divided into two equal portions, one of which is saturated with an excess of pure carbonate of soda, and evaporated to dryness over a water-bath. The other portion is evaporated in the same manner, but without the addition of the carbonate of soda. The two residues are then completely calcined in two separate porcelain capsules, treated with a little distilled water, and filtered. Each solution is then acidulated with pure nitric acid, and precipitated with an excess of nitrate of silver. The two precipitates are separately received on small filters, thoroughly washed, dried, incinerated along with their filters, and finally weighed. If the weight of the chloride of silver is the same in both cases, this will be positive proof that there was no *free* hydrochloric acid present in the original material; but if the portion saturated with the carbonate of soda yields a greater quantity of chloride of silver than the other portion, this excess of chloride must manifestly be ascribed to free hydrochloric acid.

Examination of suspected stains.—These should be treated in the manner pointed out for the other two mineral acids. Litmus-paper will indicate the presence of a free acid in the solution; and the silver test will indicate the presence of hydrochloric acid, but not its presence in the free state, exclusively. Whether the reaction is due to a chloride may be ascertained in the manner already pointed out. As hydrochloric acid is volatile, the examination of the stain should be made as early as possible; otherwise all traces may be lost.

Quantitative analysis.—Hydrochloric acid is determined quantitatively by first precipitating it as a chloride by means of nitrate of silver, and slightly heating until the whole of the chloride is deposited; the precipitate is then collected on a small weighed filter, thoroughly washed, dried, and weighed. Every 100 grains of the dried chloride correspond to 25.43 parts of anhydrous hydrochloric acid, or about 81 parts of ordinary liquid acid of sp. gr. 1.15.

SECTION IV.

POISONING BY OXALIC ACID, AND BINOXALATE OF POTASSA.

Oxalic acid, although strictly belonging to the organic acids, is conveniently discussed under the present head, inasmuch as some of its effects upon the system so thoroughly resemble those occasioned by the corrosive (mineral) acids.

This acid exists, in combination with lime and potash, in several vegetables, as the rhubarb, or pie-plant, the wood sorrel (*Oxalis acetosella*), and several others. It is a compound of carbon and oxygen with the elements of water. It is sometimes called the *acid of sugar*, because it is procured by the action of nitric acid on sugar or starch. It is a solid, white, crystalline substance, bearing a considerable resemblance to Epsom salt (sulphate of magnesia), and white vitriol (sulphate of zinc). It has an intensely sour taste, by which it may easily be distinguished from these substances. It is considerably employed in the arts, and has frequently been the cause of accidental death, from having been mistaken for Epsom salt. It has also been frequently taken for suicidal purposes, especially in England; and occasionally it has been administered homicidally. Its intensely sour taste would usually lead to its immediate detection. A case is reported in which it was administered with criminal intent in buttermilk, with fatal result.

Symptoms.—These depend very much upon the size of the dose, and the degree of concentration. When swallowed in a large dose—half an ounce to an ounce—dissolved in a small quantity of water, the effects are immediate and violent. An intensely sour taste is speedily followed by a burning sensation in the gullet, extending to the stomach, increased by pressure; there is also a feeling of constriction of the throat; vomiting soon follows, sometimes of bloody matters, but generally of a greenish-brown or black grumous matter; and if the patient survives some hours, there is purging of a similar character. The remaining symptoms are those of collapse,—extreme debility, pale and anxious countenance, cold and clammy skin, small and frequent pulse, and hurried respiration. There are also soreness of the mouth, inflammation and swelling of the tongue, painful deglutition, intense thirst, restlessness, difficulty of breathing, and distressing cough. Besides the above symptoms, there are frequently cramps and numbness of the legs and arms, acute pain in the back and head, delirium, and convulsions—symptoms which indicate the very decided action of the poison upon the *nervous* system. As in the case of other violent poisons, the above-mentioned symptoms are subject to many exceptions and anomalies. Thus, cases are reported in which pain and vomiting have both been absent; or vomiting has not occurred until emetics were administered. In a singular case referred to by Sir R. Christison (On Poisons, p. 223), a peculiar spotted eruption appeared upon the skin, and leeches applied to the epigastrium soon fell off dead, showing evidently that the poison had been absorbed into the circulation.

The general symptoms of oxalic acid poisoning are undoubtedly those of the irritants; but it may be so diluted as to lose all its irritant and corrosive properties, and yet prove fatal, from its remote specific effects upon the heart and the nerve-centres. The latter impression is evidenced in the acute pains in the back, extending down the limbs, the tetanic spasms, the numbness and tingling in the limbs, approaching to paralysis, and also the occasional narcotic effect observed. In this respect it differs from the mineral acids.

Fatal dose.—The smallest fatal quantity recorded is in the case of a boy aged sixteen years, who died in nine hours after eating about *one drachm* of the solid crystals. (Case reported by Dr. Barker, Lancet, Dec. 1, 1855; and quoted by Dr. Taylor.) The latter authority also reports a case in which a woman aged twenty-eight years died in one hour after swallowing *three drachms* of the crystallized acid. Serious, though not fatal, consequences have followed the taking of much smaller doses. As a rule, a dose of half an ounce or upwards nearly always proves fatal; although instances are on record where complete recovery has taken place after taking considerably over an ounce of the crystals.

Fatal period.—Oxalic acid, when taken in full dose and in a concentrated state, is one of the most energetic poisons known; but equal quantities do not always destroy life in the same time. Dr. Ogilvie, of Coventry, reports the most rapidly fatal case known, where death took place *within three minutes* after swallowing an unknown quantity of the acid (Lancet, Aug. 23, 1845, p. 205). Sir R. Christison calls it "the most rapid and unerring of all the common poisons." He mentions two cases—one of a young man, and another of a young lady, who survived only *ten minutes* after swallowing one ounce of the acid. The majority of the fatal cases succumb within one hour. On the other hand, numerous cases have been reported in which death did not occur for several hours, and even days. In one reported by Dr. Jackson (Boston Med. and Surg. Jour., vol. xxx. p. 17), life was prolonged until the tenth day, after swallowing an ounce of the crystallized acid in mistake for Epsom salt; and Dr. Beck (Med. Jurisp., ii. p. 439) alludes to an instance in which a woman died from the secondary effects of the poison, after several months' suffering.

Treatment.—The proper antidotes are chalk, and magnesia, or its carbonate, suspended in water or milk. These act by forming insoluble and inert earthy oxalates. The alkalies or their carbonates are inadmissible, since the alkaline oxalates are nearly as poisonous as the acid itself. After thus neutralizing the poison, free vomiting should be encouraged by the use of mucilaginous drinks. Lime-water and oil may be employed with advantage. Sometimes the stomach-pump may be employed, as when much liquid has been swallowed with the poison. In the collapse, warmth should be applied, and stimulants freely used. The secondary symptoms should be treated on general principles.

Morbid appearances.—In rapidly fatal cases the lining membrane of the mouth, throat, and gullet is usually more or less disorganized, white, and softened, but often covered with a portion of the dark-brown matter discharged from the stomach. The mucous lining of the gullet is often softened and easily detached, and its vessels much congested. The stomach contains a dark-brown mucous fluid, often acid, and having at times a gelatinous consistence. The subjacent mucous membrane will be found generally pale and softened, often without marks of a decided inflammation, if death has been rapid. It is soft and brittle, and easily removed. The small vessels are filled with dark, coagulated blood. In some cases the stomach presents very much the appearance of a case of poisoning by sulphuric acid. Sometimes the upper portion of the small intestines presents a similar appearance, especially if death has not occurred very speedily. In some cases extensive congestion of the lungs has been observed, together with a fullness of the heart and great vessels; the blood being dark-colored.

As regards *perforation* of the stomach by oxalic acid, all the authorities unite in saying that it is of rare occurrence. A few cases have been reported in which extensive softening and numerous perforations were discovered after death; but there is strong reason for believing that these conditions were due to the action of the acid upon the tissues *after death.* In protracted cases, the lining of the stomach is usually much thickened and corrugated, and much inflamed, and probably ulcerated. Congestion of the brain has been observed in at least one fatal case; but it is by no means a constant lesion. The condition of the *heart* varies: sometimes its cavities are found to be full of dark fluid blood, and at other times they are found empty, or containing merely a small clot.

Chemical analysis.—(1) *In the solid state.*—Oxalic acid occurs, when pure, in colorless, transparent, four-sided crystals, without odor, quite soluble in water, especially when hot; soluble also in alcohol, but insoluble in ether, and nearly so in chloroform. It has an intensely sour taste, which serves to distinguish it immediately from the sulphates of magnesia and zinc, which it strongly resembles in appearance, and for which (especially the former) it has been often fatally mistaken. Another characteristic mark of difference is the action of heat: if a crystal of oxalic acid be heated on platinum-foil over a spirit-lamp, it will, if pure, be completely volatilized; while the other substances leave a fixed residue.

(2) *As a liquid.*—First test its acidity by litmus-paper; if acid, evaporate a few drops to dryness: oxalic acid will leave a *crystalline* residue, consisting of long and slender prisms. The solution is tested, (*a*) by *nitrate of silver:* this yields an abundant white precipitate (oxalate of silver), which is distinguished from the *chloride* and the *cyanide* by being immediately soluble in *cold* nitric acid (the chloride is not soluble even in boiling nitric acid, and the cyanide is insoluble in the cold acid). If the precipitated oxalate of silver be thoroughly dried, and heated on platinum-foil, it is entirely dissipated in white vapor, with slight puffs or detonations: this does not occur with the chloride or the cyanide. (*b*) *Sulphate of lime.* Any solution of lime will precipitate oxalic acid, but the sulphate is preferable, because it is not acted upon by many substances which precipitate other salts of lime. As the sulphate of lime is not very soluble, it should be added in considerable quantity to the suspected oxalic solution. A fine white precipitate (oxalate of lime) is slowly formed, which is immediately soluble in nitric acid, more slowly in hydrochloric acid (in excess), and insoluble in the vegetable acids—acetic,

citric, and tartaric. The addition of ammonia or potassa will enable the sulphate of lime to precipitate the oxalic acid more abundantly; but, as this might possibly lead to complications in a medico-legal case, it had better not be employed. The only objection that can be urged against the sulphate of lime test is, that it will precipitate even acid solutions of baryta, strontia, and lead; but this is readily obviated by the fact that the sulphates of these bodies are entirely insoluble in nitric acid; while oxalate of lime is very soluble in it. According to Prof. Wormley, the precipitate from sulphate of lime shows under the microscope oval granules, while that from the chloride of calcium exhibits octahedral crystals and small plates, somewhat larger than the former. (*c*) *Chloride of barium*, *nitrate of strontia*, and *acetate of lead* all precipitate oxalic acid in the form of white crystalline deposits; but these tests are liable to more fallacies than the two former ones, and are, therefore, of inferior value. (*d*) *Sulphate of copper* causes a faint bluish-white, or greenish-white, precipitate, which is insoluble in acetic acid, and almost so in nitric acid. This reagent also precipitates the carbonates and phosphates, and is decomposed by various kinds of organic matter, yielding a somewhat similar precipitate; but all these deposits are distinguished from the oxalate of copper in being readily soluble in nitric and hydrochloric acids.

Detection in organic mixtures.—Although oxalic acid is not decomposed by organic mixtures, yet these would be very apt to cause precipitates with two of the tests, viz., nitrate of silver and sulphate of copper. Hence these reagents cannot be relied upon in organic solutions. If the solution is strongly acid, and contains solid matters suspended, it should first be mixed with distilled water, if necessary, and be digested at a moderate heat for some time, then cooled and filtered, and the filtrate concentrated by evaporation, and again filtered. A small quantity may now be tested with sulphate of copper, as a trial test. If this causes a bluish-white precipitate not readily soluble in strong nitric acid, it is due most probably to oxalic acid. If the filtered solution is very strongly acid, it may be allowed to crystallize by evaporation; and the crystals removed and washed, and dissolved in water, will respond to the usual tests. If the crystals are very highly colored, they should be redissolved in warm water, and recrystallized.

Should, however, the solution prove to be only faintly acid, and mixed with much organic matter, after proper warming, filtering, and concentration, it should be treated with acetate of lead in excess, which will precipitate the whole of the oxalic acid as oxalate of lead. The precipitate should be collected and washed with water acidulated with acetic acid; and afterwards with pure water. The moist precipitate is then diffused in water, through which a stream of sulphuretted hydrogen is passed until all the lead is precipitated as a sulphide; along with this, most of the organic matter will also be thrown down. By filtering, a clear acid liquid will be obtained: this should be moderately heated, to expel any excess of sulphuretted hydrogen; and on evaporation, the characteristic crystals of oxalic acid may be obtained. These should be purified by dissolving in alcohol, and subsequently in water. The oxalate of lead obtained as above may likewise be decomposed by boiling with dilute sulphuric acid; sulphate of lead is formed, which is separated by filtration from the oxalic acid solution. Neutralize cautiously with ammonia; filter again, to remove any oxalate of lead resulting from the sulphuric acid; then apply the usual tests to the clear solution. Of the two processes for decomposing the oxalate of lead, the former is the more reliable for obtaining the acid in the crystalline form.

The processes above mentioned will yield the same results whether oxalic acid exist in the free state, or combined with an alkaline or earthy base: consequently, they do not serve to determine this question. Now, in the contents of the stomach, it will probably happen that the poison has been completely neutralized by the antidote administered—lime or magnesia: hence the acid would exist as an oxalate of one or other of these bases. In the latter case, the matters may have a neutral reaction; the suspected solids should then be carefully collected in a dish and thoroughly washed with warm water, and the liquid decanted. If this liquid is acid, it should be reserved; but if not, it may be thrown away. The solids should then be diffused in a little pure water, and boiled with a proper quantity of pure carbonate of potassa, for about half an hour; the loss by evaporation being supplied by additions of pure water. A double decomposition will give rise to the soluble oxalate of potassa and the insoluble carbonate of lime, or of magnesia. After cooling and filtering, the filtrate is treated with excess of acetic acid, then precipitated by acetate of lead; the precipitate is then decomposed by means of sulphuretted hydrogen; the sulphide of lead is separated by filtration, the clear liquid is evaporated, and tested as before mentioned.

In case of the oxalic acid existing in combination with potash, as after partaking of *sorrel*, an approximative, though not accurate, method of distinguishing between the free and the combined acid, is to evaporate the original acid liquid to dryness on a water-bath, and extract the residue with very strong alcohol, which dissolves the *free* acid if present, but leaves undissolved most of the oxalate. The filtered alcoholic solution is now evaporated to dryness on a water-bath, the residue dissolved in a small quantity of water, filtered, concentrated, and tested as usual. The portion undissolved by the alcohol is stirred with distilled water, filtered, and the filtrate examined in the usual manner. The above is Orfila's process for separating the free acid from its association with an alkaline oxalate. But Professors Christison and Taylor very properly object to this being considered a safe and reliable process in medico-legal cases, inasmuch as the strongest alcohol will always acquire a slight acid reaction when digested on binoxalate of potassa; and hence an analyst would be deceived if he relied solely on this process, and might be led to pronounce that to be free oxalic acid, when in reality it was due only to the alkaline oxalate, accidentally taken by the deceased, as in sorrel-soup!

This leads us to notice more particularly the fact already adverted to, that certain articles of food—more particularly the rhubarb, or pie-plant, and the sorrel—contain a notable quantity of oxalic acid, not, however, free, but combined with lime or potash. It might possibly happen that in a case of poisoning, the discovery of merely a small quantity of the acid by the usual tests would be ascribed by the defense to one of these vegetables that had been eaten by the deceased. The answer is very obvious: in a case of true oxalic acid poisoning, the symptoms and the morbid lesions are of such unequivocal character that, unless these can be clearly proven, the discovery of a minute quantity of the acid is no evidence of poisoning; but, on the other hand, if the peculiar symptoms and morbid lesions are present, then the obtaining of only a small quantity of the acid should not negative the charge of poisoning. Sir R. Christison regards oxalic acid as one of the few poisons "of whose operation distinct evidence may sometimes (though certainly not always) be found in the symptoms." "If," says he, "a person immediately after swallowing a solution of a crystalline salt, which tasted purely and strongly acid, is attacked with burning in the throat, then with burning in the stomach, vomiting, particularly of bloody matter, imperceptible pulse, and excessive languor, and dies in half an hour, or, still more, in twenty, fifteen, or ten minutes, I do not know any fallacy that can interfere with the conclusion that oxalic acid was the cause of death. No parallel disease begins so abruptly, and terminates so soon, and no other crystalline poison has the same effects." (On Poisons, p. 226.) To this Prof. Guy adds: "The post-mortem appearances are scarcely less characteristic. The wrinkled and corroded gullet, the pale, shriveled, and partially detached mucous membrane of the stomach, the dark veins ramifying on its surface, and the dark-brown grumous matter which fills its cavity, point strongly to the action of a powerful corrosive poison, while the absence of the colored spots on the skin would preclude the supposition of the effect being due to either of the mineral acids." (Guy's For. Med., p. 595.)

Is oxalic acid a normal constituent of the body?—It is extremely doubtful if this acid, in the free state, ever exists in the animal system. Liebig and others suppose that one of the products of the ultimate oxidation of uric acid in the human system is oxalic acid: this change can be readily exhibited in a chemical formula, but no one, we believe, has ever demonstrated its presence in the blood or in the tissues. Orfila, Christison, and Taylor all failed to detect it in the blood or in the organs, in cases where very large doses had proved fatal, and even where it had been previously injected into the femoral vein of an animal which died in thirty seconds (Christison, On Poisons, p. 219). Nevertheless, its presence in the circulation, in some form, would seem to be shown by the fact, already cited, that in two instances on record, leeches applied to persons poisoned by this acid speedily fell off dead. Moreover, Orfila states that he succeeded in detecting the acid in the urine of a person poisoned with it (Toxicol., i. 190). The *oxalate of lime* is frequently found in the urine as the result of disease, but never as a normal constituent. It also occurs as one variety of calculus in the bladder (*mulberry calculus*). In case of poisoning, the oxalic acid is, no doubt, excreted by the kidney as oxalate of lime, which can easily be detected in the urine by the microscope, in the well-known form of octahedral crystals. It must, however, be remembered that these same crystals will be found in the urine of persons who have partaken of food containing the oxalate of lime,—such as the rhubarb or the sorrel-plant.

In examining the urine for *free* oxalic acid, or a soluble oxalate, a little acetic acid should first be added, and then it should be evaporated to about one-fourth its bulk, and filtered: to the filtrate an excess of acetate of lead is added, and the resulting oxalate of lead, after thorough washing, is decomposed by sulphuretted hydrogen, and the filtered solution tested as above.

Quantitative analysis.—Oxalic acid from pure solutions is best estimated as *oxalate of lead*. The solution is first treated with a little acetic acid, and then with a solution of acetate of lead in excess; and the precipitate collected on a filter of known weight, completely washed, and dried at 212°; and then weighed. Every 100 parts of the oxalate of lead thus obtained, represents 42.5 parts of the crystallized acid.

If the acid has been precipitated as *oxalate of lime*, this should be thoroughly washed and dried, and then exposed for a few minutes to a dull red heat, by which it is converted into *carbonate of lime;* every 100 parts of which correspond with 126 parts of crystallized oxalic acid.

Examination of stains.—Oxalic acid does not corrode cloth and other textures like the mineral acids; but it very slowly produces orange-colored spots, with a red margin, on black cloth, differing in this respect from the other vegetable acids. Proofs of its presence in these stains may be obtained by soaking them in hot water, and applying the proper tests to the solution. The acid is sometimes used to remove writing-ink in cases of forgery; but, usually, traces of iron, existing in the ink, are left upon the paper, which can easily be detected by wetting it with a solution of ferrocyanide of potassium, which will turn it blue.

BINOXALATE OF POTASSA (*Salt of sorrel.—Essential salt of lemons*).—This salt is much used in the arts for bleaching, and for removing ink-stains: it is sold under the absurd name of *essential salt of lemons.* Its poisonous properties are almost as violent and active as those of oxalic acid. Half an ounce produced death in a lady, in *eight minutes*, after violent pain and convulsions (Jour. de Chim. Méd., 1842, p. 211). In another case, a teaspoonful of this salt was taken for three successive mornings, causing severe vomiting; an hour after the third dose, the patient died (Ann. d'Hyg. Pub., 1842, xxvii. p. 422). A case is reported of a young lady, aged twenty, who recovered after swallowing an ounce of this salt dissolved in water. The symptoms were vomiting, a scalding sensation of the throat and stomach, great depression, faintness, cold and clammy skin, and feeble pulse. There were also great dimness of vision, redness of eyes, and dilatation of the pupils (Med. Gaz., xxvii. p. 480).

Chemical analysis.—This salt commonly occurs in crystals; it is not very soluble in cold water; much more so in hot water; taste very acid. It is distinguished from oxalic acid by—(1) its plumose crystalline form, seen by evaporating a few drops on a glass slide. (2) By heating a portion on platinum-foil: an ash is left (carbonate of potash), while oxalic acid is entirely dissipated. It may readily be distinguished from the bitartrate of potash (cream of tartar), for which it has been fatally mistaken, by the latter not being precipitated by the sulphate of lime. Lime-water will also serve to distinguish them: it throws down a white precipitate with each, but the tartrate of lime is immediately redissolved by tartaric acid, while the oxalate of lime remains insoluble. Common writing-ink immediately loses its color on being warmed with binoxalate of potassa, but it is unaffected by the bitartrate (Taylor). This salt is a natural constituent of the sorrel, which is used considerably in France as an article of food.

SECTION V.

POISONING BY TARTARIC ACID.—CITRIC ACID.—ACETIC ACID.

These acids, although not usually classed among poisons, are nevertheless capable of destroying life, if taken in large doses, and in concentrated strength.

TARTARIC ACID.—This is the acid of grapes, and of a number of other fruits. It occurs in large, oblique, rhombic, colorless crystals; quite soluble in water; less so in alcohol. It may be recognized by the following characters: (*a*) Its solution gives no precipitate with nitrate of silver, which distinguishes it from oxalic acid; (*b*) on evaporation of a few drops on glass, it yields plumose crystals; (*c*) it precipitates a solution of potash or its salts, when of moderate strength, as a granular powder (cream of tartar): this precipitate is facilitated by stirring with a glass rod, or by the addition of a little alcohol; (*d*) when the powdered acid is heated on platinum-foil, it burns with a pale reddish-colored flame, evolving a peculiar odor, and leaving an abundant residue of carbon.

Several cases of poisoning by this acid are on record. One is mentioned by Dr. Taylor, in which a young man swallowed, by mistake, *one ounce* of tartaric acid dissolved in half a pint of water. He immediately experienced very violent symptoms, such as burning in the throat and stomach, the patient comparing his sensations to being on fire. Vomiting set in, and, although the proper antidotes were administered, he died in nine days. On inspection, the whole alimentary canal was found violently inflamed. Another case of fatal poisoning by this acid was published by M. Devergie (Ann. d'Hyg., 1851, ii. p. 432); and two cases—one of them fatal—by M. Tardieu (*loc. cit.*, p. 253).

The proper *treatment* is free vomiting, to be followed by the exhibition of the alkaline carbonates, chalk, or magnesia.

The resulting inflammation is to be treated on general principles.

From *organic mixtures*, or *contents of the stomach*, the acid may be extracted by the process of *dialysis* (*ante*, p. 113), or by digestion in alcohol, which will dissolve it, and afterwards deposit it on evaporation.

Citric Acid.—This is the acid of lemons. Experiments on animals would seem to show that it is a more powerful poison than tartaric acid. We are not aware that any fatal case of poisoning in the human subject has been reported.

The proper treatment to be pursued is the same as that pointed out for tartaric acid.

Acetic Acid.—This acid is found in the shops under several forms. *Common vinegar* is a very dilute impure variety; it contains on an average from four to five per cent. of acetic acid. The vinegar of commerce frequently contains sulphuric acid as an impurity.

Pyroligneous acid, or *wood vinegar*, obtained by the destructive distillation of wood, contains from twenty-five to fifty per cent. of the strong acid. A third form is the concentrated, or pure acetic acid. This is a colorless, pungent liquid, possessing a peculiar odor, acting upon the animal tissues as a violent irritant, or even as a corrosive.

Orfila reports the case of a young woman who was found dying upon the highway: she suffered from convulsions, complained of great abdominal pain, and died in a short time. On inspection, the stomach was found of a deep-black color, the vessels being gorged with dark coagulated blood. The examination showed that death had been caused by strong acetic acid, which had been taken probably as an abortive. (Toxicologie, ii. p. 198.)

The *treatment* is similar to that mentioned for tartaric acid.

Analysis.—Acetic acid in organic mixtures may generally be recognized by its peculiar odor: if this does not suffice, the mixture should be distilled in a glass retort, the distillate neutralized with carbonate of potassa, and evaporated. The resulting *acetate of potassa*, when distilled with sulphuric acid, yields the pure acetic acid.

CHAPTER XII.

POISONING BY THE ALKALIES AND THEIR SALTS; ALSO BY THE EARTHY SALTS.

SECTION I.

POISONING BY THE ALKALIES.—POTASSA.—SODA.—AMMONIA.

THE effects of the strong alkalies upon the animal system are very similar to those of the mineral acids: like the latter, in their concentrated state they are powerfully corrosive, attacking the tissues by virtue of their chemical affinities, and causing complete destruction or disorganization of the parts with which they come in contact. Although they are in common use for domestic purposes, they are very seldom taken or administered as poisons, except accidentally.

Pure *caustic* potassa and soda are kept almost exclusively in chemical laboratories. The substances generally known as potash and soda are the impure *carbonates;* they are sold under the names of *potash* and *pearlash*, and *soda-ash*, and they contain a varied proportion of the true alkali. They are, however, powerfully caustic in their effects, and the remarks that follow have reference to them as well as to the *pure* alkalies. Their general effects upon the system are so very similar that these may conveniently be considered together. Potassa and soda are usually spoken of as the *fixed alkalies;* whilst ammonia is termed the *volatile alkali.* The latter, together with its salts, is dissipated by heat; while the former remain fixed. By this means they are easily distinguished from each other.

Symptoms.—When swallowed in a concentrated solution, a nauseous, acrid taste is *immediately* perceived. This is accompanied by a burning sensation in the throat and gullet, which extends to the stomach, and very soon changes to acute and violent pain, which is increased by pressure, and followed by excessive vomiting of mucous matters mixed with blood. Sometimes the very acrid and nauseous taste of the substance causes it to be immediately rejected from the mouth without being swallowed. Purging of stringy mucus mixed with blood soon comes on. There is difficulty of swallowing, with hoarseness of voice, and cough. Great muscular prostration soon occurs, with small and frequent pulse, clammy perspiration, and other evidences of collapse. If death does not come on very soon, there may be sloughing of the fauces, with increased difficulty of swallowing, constant vomiting of bloody mucus, and tenesmus. Stricture of the œsophagus is a very common result, as in the case of poisoning by the mineral acids. The patient may live for months, suffering greatly, and at last perish from starvation, owing to an inability to swallow food.

The effects of swallowing a strong solution of *ammonia* are similar to those occasioned by the fixed alkalies, except that they are occasionally more severe and rapid in their action. Cases are recorded where death ensued in six hours; and one is quoted by Sir R. Christison (On Poisons, p. 194), in which a quantity of liquid ammonia poured into the mouth of a man who had been bitten by a mad dog, destroyed life in *four minutes.* In this case, it is highly probable that the fatal event was hastened by the irritant impression of the vapor upon the organs of respiration. Dr. Taylor records the case of a gentleman who died in *three days* after swallowing a solution of ammonia by mistake. The *vapor* of ammonia has frequently proved fatal from being accidentally or incautiously inhaled. If the patient survives the primary effects of poisoning from solution of ammonia, he is more likely to recover ultimately, than from poisoning by potash or soda.

Fatal dose.—As in the case of the corrosive acids, the fatal result in poisoning by the alkalies depends rather upon the degree of concentration, than upon the mere quantity taken. The smallest fatal dose recorded is in a case mentioned by Dr. Taylor (On Poisons, p. 328), where an ounce and a half of the common solution of potash of the shops, containing about *forty grains*, proved fatal to an adult in seven weeks.

Several other cases have occurred in which *half an ounce* caused death. In each of these instances, death was due to the secondary effects of the poison.

Strong solution of *ammonia* has proved fatal in the dose of *two drachms.* But instances of recovery from taking this alkali are more frequent than from the others. Cases are on record in which persons have survived after swallowing more than an ounce of liquor ammoniæ.

Treatment.—In all cases of poisoning by the alkalies or their carbonates, the prompt use of some of the mild vegetable acids is indicated: vinegar and water, or lemon-juice, answers well. Large quantities of olive oil, or of other bland oil, are also useful: these act by converting the alkali into a soap. Copious draughts of milk have also been recommended. The stomach-pump should not be used, on account of the risk of perforating the œsophagus. In poisoning by vapor of ammonia, the inhalation of the vapor of acetic acid would be beneficial. The resulting inflammatory symptoms should be treated upon general principles.

Morbid appearances.—In acute cases, the lining membrane of the throat and gullet is softened and corroded, the œsophagus, stomach, and intestines are inflamed, their mucous membrane abraded more or less in patches; sometimes there are extravasations of disorganized blood upon the walls of these organs, giving them a blackish appearance. As in the case of the mineral acids, we may sometimes find large portions of the epithelium detached from the mouth, gullet, and stomach. In chronic cases, besides the above appearances, there is usually great contraction of the œsophagus and stomach. The walls of the stomach are also sometimes much thickened, and the lining membrane completely destroyed. Sometimes the larynx and bronchi are implicated: this is particularly apt to be the case in poisoning by ammonia.

In the case of poisoning by ammonia quoted above, which proved fatal in three days, the lining membrane of the trachea and bronchi was softened, and covered with layers of false membrane, whilst the larger bronchi were completely obstructed by casts of this membrane. The mucous membrane of the gullet was softened, and the lower end of the tube completely destroyed. The anterior wall of the stomach contained an aperture about an inch and a half in diameter, through which the contents of the organ had escaped. The blackened and congested appearance of the lining membrane somewhat resembled that seen in poisoning by sulphuric or oxalic acid. The immediate cause of death was asphyxia, resulting from inflammation of the air-tubes. No trace of ammonia could be detected by chemical analysis, after death.

Chemical properties of the alkalies.—The alkalies as a class are distinguished from all other bodies by their not being precipitated either by sulphuretted hydrogen, sulphide of ammonium, or carbonate of ammonia. From their *carbonates* they are easily distinguished by the action of an acid, as hydrochloric, which causes effervescence with the latter (from the escape of carbonic acid), but not with the former.

Corrosive sublimate produces with the fixed alkalies a *yellow* precipitate, insoluble in an excess of the alkali; with ammonia, it causes a *white* precipitate, soluble in an excess of the reagent.

All the alkalies possess in common the properties of neutralizing acids; of browning a solution of turmeric; and of restoring the blue color to reddened litmus. They unite with oils and fats to form soaps.

The special chemical properties of the three alkalies will now be considered separately.

Chemical analysis of Potassa and its carbonate.—Caustic potassa, when pure, occurs generally in the form of thin sticks, nearly white. It is very deliquescent, and if exposed to the air it absorbs carbonic acid, and is thus partially converted into the carbonate. It is very soluble in water, and also in alcohol: the latter property enables us to separate it from the carbonate, which is insoluble in alcohol. The impure *carbonate* (potash and pearlash) occurs in grayish-white masses: like the former, it is highly alkaline; it has an unctuous feel, is very acrid to the taste, and is freely soluble in water. The *pure* carbonate (salt of tartar) occurs in white granules: its properties are similar to those of the others.

A solution of potash, or of its compounds, is distinguished (1) by *bichloride of platinum*, which precipitates from it, if not too dilute, the yellow double chloride of platinum and potassium, which soon assumes the form of beautiful octahedral crystals. These are well shown with a drop or two of the reagents under the microscope. According to Prof. Wormley (Micro-Chem. of Pois., p. 75), one five-hundredth of a grain of potash in the form of chloride, in one drop of water, will exhibit this test satisfactorily.

Fallacy.—Bichloride of platinum will yield a similar yellow crystalline precipitate with the salts of ammonia: the absence of these must therefore first be secured before the presence of potassa can be established. This may easily be done by heating a little of the suspected liquid in a test-tube along with hydrate of lime, or caustic potash: if ammonia be present, the strong odor of this alkali will be perceived. Or the yellow precipitate may be dried and heated to redness: the potassium compound will be resolved into chloride of potassium and metallic platinum; while the ammonium compound will leave only a residue of metallic platinum. The action of nitrate of silver upon the solutions of these two residues will at once show the difference, by yielding a precipitate (chloride of silver) with the former, but none with the latter.

(2) *Tartaric acid*, or the *tartrate of soda*, throws down from a rather strong solution of potassa and its salts a white crystalline precipitate of *cream of tartar*. If the potash solution is dilute, the precipitate is retarded; but it may be hastened by stirring with a glass rod, when it shows itself in streaks upon the sides of the tube; it is also facilitated by the addition of alcohol. The precipitate is soluble in the mineral acids, and in the free alkalies and their carbonates. Hence, when a *salt* of potassa, *e.g.* the nitrate, is treated with free tartaric acid, unless the former be in strong solution, the nitric acid which is set free by decomposition may prevent the precipitation of cream of tartar. On this account it has been recommended to employ the *acid tartrate of soda* in preference to the free tartaric acid, as the reagent. This is made quite readily by dividing a strong solution of tartaric

acid into two equal parts; neutralizing one of them with pure carbonate of soda, and then adding the other.

In using the above test, the absence of ammonia should first be proved, since this alkali yields a similar precipitate with the reagent.

(3) It is readily distinguished from soda by neutralizing with dilute nitric acid, and evaporating on a slip of glass: the crystals exhibit the characteristic appearance of nitrate of potassa—long and slender prisms. Soda, treated in the same manner, yields rhombic plates.

(4) Heated on a clean platinum wire in the reducing flame of the blowpipe, potassium compounds impart a *violet* color to the flame. This color may, however, be completely disguised if there is the smallest portion of soda present, which would give a bright-yellow color to the flame.

Other tests have been proposed, but they are of inferior value. These are *carbazotic or picric acid, perchloric acid, and hydrofluosilicic acid.*

The *spectrum process* will detect the minutest portion of any potassium compound, producing two distinct and well-marked lines or bands—one red, and the other indigo-blue. (See Quar. Jour. of Chem. Soc., Oct. 1860.)

In *organic mixtures.*—The solution will possess a strong alkaline reaction, unless previously neutralized, and a soapy feel: the absence of ammonia should first be established (*ante*, p. 179). The mass may next be evaporated to dryness, and then heated to redness in a capsule, to char the organic matters. It is then to be digested in water, and filtered: the alkali will be recovered in the form of carbonate. If it is desired to separate any free alkali from its carbonate, in the original mass, this may be effected by evaporating carefully to dryness, and digesting the cooled residue in absolute alcohol, which will dissolve out the alkali, and leave the carbonate and other salts, together with most of the organic matters, untouched. The alcoholic solution is now concentrated by evaporation, neutralized by hydrochloric acid, and tested as above; or, if much organic matter is present, the alcoholic residue should be treated as above mentioned, and the residue dissolved in water; and the solution examined in the ordinary manner.

Quantitative estimate.—Potash is estimated quantitatively by precipitating it as chloride of platinum and potassium (see *ante*, p. 179). The precipitate should be washed with strong alcohol, and then collected on a weighed filter, washed again, dried, and weighed. Every 100 parts by weight of the double salt thus obtained represent 22.5 parts of caustic potassa, or 28.25 parts of anhydrous carbonate.

Chemical analysis of Soda and its carbonate.—The general appearance and characters of *caustic* soda are very similar to those of caustic potassa. The impure *carbonate* (soda-ash) occurs in granular lumps. The pure carbonate is in the form of large colorless crystals; it contains much water of crystallization. They effloresce, on exposure to the air, and are quite soluble in water, but not in alcohol. All the soda compounds are soluble in water, except the *antimoniate*, *carbazotate*, and *tartrate*.

(1) *Antimoniate of potassa* will throw down from a strong solution of a soda salt a white, crystalline antimoniate of soda. The presence of the carbonate of potassa prevents the above precipitation, in consequence of dissolving it. As many of the metallic salts are likewise precipitated by this reagent, their absence should first be ascertained before concluding that the precipitate is a soda compound.

(2) *Polarized light*, as first suggested by Prof. Andrews (Chem. Gaz., x. p. 378), affords a beautiful method of distinguishing soda and its compounds from potassa. Bichloride of platinum, which precipitates the latter, has no such effect upon the former alkali, although it forms with it the double chloride of platinum and sodium. The latter salt possesses the depolarizing action; but the former does not. The method of applying the test is as follows. A drop of the alkaline solution (converted into the chloride) is placed on a glass slide, and a drop of the solution of bichloride of platinum is added, avoiding any excess. The mixture is evaporated by a gentle heat until it begins to crystallize; it is then placed in the field of a microscope furnished with a good polarizer. On turning the analyzer till the field becomes perfectly dark, and carefully excluding the light laterally, the crystals exhibit a beautiful display of colors; whilst

if no soda be present, but only the bichloride of platinum, or potassa, no effect is produced. Prof. Andrews states that this test is so extremely delicate that he obtained by it a distinct reaction from a quantity of chloride of sodium representing only about one eight-hundred-and-twenty-five-thousandth part of a grain of anhydrous alkali.

Prof. Wormley confirms the delicacy of the above test (Micro-Chem. of Poisons, p. 86). He recommends to evaporate the mixture on the glass spontaneously, and not to use heat: by this means, larger crystals of the double salt are observed.

(3) Both carbazotic and tartaric acids will cause crystalline precipitates in *concentrated* solutions of soda, which are quite distinct in appearance from the corresponding salts of potassa. These tests, however, are inferior in value to the preceding ones.

(4) Heated on a clean platinum wire in the blowpipe flame, it imparts to it a bright-yellow color. The *spectrum* process also yields a very characteristic yellow band.

The mode of detection of soda in *organic mixture* is the same as that employed in the case of potassa.

Chemical analysis of Ammonia and its salts.—Solution of ammonia (*Aqua* or *Liquor Ammoniæ*) is a colorless limpid fluid, having a very strong pungent odor, an acrid alkaline taste, and a strong alkaline reaction. When heated, gaseous ammonia is given off; and when evaporated to dryness, it leaves no residue. It gives to a solution of sulphate of copper a characteristic purple color.

The *salts* of ammonia are colorless, and volatilize when heated. They are mostly soluble in water. Heated with the fixed alkalies, or with hydrate of lime, they readily decompose, evolving the characteristic odor of ammonia. If the experiment be performed in a small test-tube, and a piece of moistened reddened litmus-paper be placed within the tube, the disengaged ammoniacal gas will very soon restore the blue color to the paper; and if a glass rod holding a drop or two of hydrochloric acid be held over the mouth of the tube, the characteristic white fumes of chloride of ammonium will be observed.

The above methods are amply sufficient to demonstrate the presence of ammonia or its salts. Several other corroborative tests may, however, be employed. These are *bichloride of platinum*, which gives with ammonia a precipitate (chloride of platinum and ammonium) very similar in color and crystalline form to that obtained from potash. The mode of distinguishing between them has been pointed out above (p. 179). *Tartaric acid* and the *acid tartrate of soda* yield with solutions of ammonia a precipitate almost identical in appearance with that given by potassa. *Carbazotic acid* likewise throws down from solutions of ammonia a precipitate of yellow crystals, differing, however, in appearance from the corresponding precipitates with the fixed alkalies.

Carbonate of ammonia is at once distinguished from the pure alkali, by its effervescing with acids.

Detection in organic mixtures.—Unless present in very minute quantity, free ammonia will always be made evident by its odor, and by its alkaline reaction. Both ammonia and its carbonate may be separated from organic mixtures by distillation at a moderate heat, and collecting the vapors by means of a small bent delivery-tube beneath a small quantity of water contained in a well-cooled receiver. The solution of ammonia thus obtained can be tested in the ordinary manner.

If there is no evidence of ammonia in the distillate after conducting the process for some time, this would be proof that there is no free alkali or its volatile salt present. It might, however, be in the retort in the form of one of the more fixed salts. To ascertain this, the contents of the retort are to be treated with strong alcohol, which will coagulate the organic matter; filter the solution, and distill it along with some hydrate of lime, or solution of potassa. Any ammoniacal salt will now undergo decomposition, and free ammonia will come over, and may be received under water, or in a dilute solution of hydrochloric acid.

It should be remembered that all animal matters in a state of decomposition give out ammonia: hence it may be impossible, under such conditions, to decide upon the true source of the alkali. In a medico-legal case, we would be materially aided in the investigation by the *quantity* of the poison recovered, and also by the symptoms and the post-mortem appearances.

Quantitative determination.—This is effected by precipitating the whole of the ammonia as a double chloride of ammonium and platinum; and proceeding as in the case of potassium (*ante*, p. 181). Every 100 parts of the double salt represent 7.62 parts of pure ammonia.

SECTION II.

POISONING BY THE ALKALINE AND EARTHY SALTS.—NITRATE OF POTASSA.—BITARTRATE OF POTASSA.—SULPHATE OF POTASSA.—ALUM.—CHLORINATED POTASSA AND SODA.—SALTS OF BARYTA.

NITRATE OF POTASSA (*Nitre. Saltpetre*).—This well-known salt is extensively used in the arts; and also in medicine in small doses, as a sedative and diuretic. It occurs in long six-sided prismatic crystals (*common saltpetre*); and in white globular masses and cakes, made by fusion (*sal prunelle*). Its taste is peculiar, cooling but sharp; it is very soluble in water. It deflagrates when thrown upon hot coals, and yields nitrous fumes by the action of sulphuric acid. The crude salt of commerce contains chloride of sodium: hence, when acted upon by sulphuric acid, it may give off chlorine, or hydrochloric acid gas.

This salt constitutes an exception to the general effect of neutralization on the local irritants. Both its acid and its alkali are simple, though powerful, irritants; yet the compound salt, although inferior, is still energetic. But its action is something more than merely irritant, since experience shows that it often causes symptoms which indicate its influence upon the nervous centres.

In small doses, as five to ten grains, it is a valuable medicine. Its poisonous effects appear to be owing to its degree of concentration, rather than to the mere quantity of the salt. Thus, it has often been administered medicinally in doses of half an ounce to two ounces in the twenty-four hours, diffused in large quantities of barley-water, or other diluent, without any injurious effect. There can be no doubt, however, that a like quantity taken at one dose, and in a concentrated form, would cause most serious, and even fatal, results.

Symptoms.—The experiments of Orfila and others upon animals show that this salt has a twofold action,—the one irritating, and the other narcotic. Death was preceded by giddiness, slight convulsions, dilated pupil, insensibility, and paralysis. In man, the usual symptoms are vomiting, sometimes of blood, violent burning pain in the throat and stomach, followed by coldness of skin, weak and frequent pulse, bloody stools, collapse, and death. Nervous symptoms, such as tremors, spasms, loss of speech and sensation, and hallucinations, are sometimes exhibited. Occasionally, when very large doses have been taken, the local symptoms have been comparatively slight, whilst the impression on the nervous centres is more decided.

Fatal dose.—In a case recorded by Dr. Beck, a dose of saltpetre, taken in mistake for Glauber's salt, proved fatal to an aged man in half an hour. Orfila quotes the case of a lady who swallowed an ounce of nitre by mistake for other salts. In a quarter of an hour she suffered from nausea, vomiting, and purging; and the muscles of the face were convulsed. The pulse was weak, the respiration feeble, and the limbs cold. There was a sense of burning and severe pain in the stomach. She died in *three hours* after taking the dose. On inspection, the stomach was found highly inflamed, and the mucous membrane detached in various parts. Near the pylorus, the inflammation was of a gangrenous character. (Toxicologie, i. p. 355.) Dr. Taylor mentions a case of a man who took nearly an ounce and a half of nitre by mistake for salts. Severe pain of the abdomen, with vomiting, followed, but no purging. He died in *two hours* after taking the dose. The post-mortem examination revealed a condition of the stomach similar to that of the preceding case. (On Poisons, p. 306.) In other instances, the symptoms were those of the most violent cholera, but were more protracted than in the cases above mentioned.

The largest dose recorded to have been taken is mentioned in Wharton and Stillé's Medical Jurisprudence, 1873, ii. p. 334. A German by mistake swallowed *three ounces and a half* of the salt. He complained of but slight pain or sense of heat in the stomach, and was purged three times within three or four hours. About five hours after taking the nitre, he suddenly fell out of his chair, and expired. There was no post-mortem examination. In this case there was a remarkable absence of the usual signs of local irritation and inflammation. The poison seems to have acted after the manner of a shock, or, as has been suggested, by destroying the vitality of the blood. The rigor mortis was very imperfect, and the countenance and lips retained their life-like appearance, to a remarkable degree, for three days after death.

Numerous instances have occurred of recovery after taking large doses of saltpetre, varying from half an ounce to two ounces. In some of these cases the sufferings of the patient were very severe, and lasted for several months.

Post-mortem appearances.—The effects of this substance in a poisonous dose are observed chiefly in the stomach, which is usually found much inflamed, mottled with dark-colored patches, and the mucous membrane partially detached. The small intestines have exhibited a similar appearance. The contents of the stomach are sometimes deeply tinged with blood. Perforation has been observed in one instance.

Treatment.—There is no chemical antidote. Free vomiting should be encouraged, and mucilaginous drinks copiously taken. The symptoms of depression should be met with the use of stimulants, opium, camphor, etc.; antiphlogistics may be required to combat the inflammatory symptoms.

Chemical analysis.—(See NITRIC ACID, *ante*, p. 152.) The researches of Wöhler prove that nitre is absorbed into the circulation, and eliminated by the *urine*, in which secretion it has been detected by chemical reagents. Orfila states that he detected it in the liver, spleen, kidneys, and urine of animals poisoned by it. (Ann. d'Hyg., 1842, ii. p. 434.)

BITARTRATE OF POTASSA (*Cream of Tartar*).—This salt, although not generally considered poisonous, has destroyed life in at least one instance, where an adult male took about two ounces, and died in forty-eight hours, with symptoms and post-mortem appearances much resembling those caused by nitrate of potassa.

It is usually found in the shops as a white powder: it has an agreeable acid taste, and is sparingly soluble in water.

When heated on platinum-foil, it is converted into carbon and carbonate of potassa: the latter is recognized by its effervescing with acids. When mixed with organic liquids, owing to its sparing solubility, it is generally found as a sediment.

The *treatment* is similar to that recommended for poisoning by nitrate of potash. A dilute solution of the bicarbonate of potassa may be advantageously given, as it reduces the bitartrate to the condition of the neutral tartrate—a harmless salt.

SULPHATE OF POTASSA.—This salt, like the two former ones, has given rise to violent and fatal symptoms, when taken in large doses. Ten drachms given in divided doses proved fatal to a French lady, within a week of her confinement, in *two hours*, with the symptoms and post-mortem appearances of an irritant poison. It has occasionally been used in Europe, particularly in France, as an abortive; and has proved fatal in this way. The *fatal dose* may be considered to be from one to two ounces.

Sulphate of potash has been found to be occasionally contaminated with arseniate of potash. M. Bussy found this poison in a sample supplied by a wholesale house of Paris (Pharm. Jour., May, 1872, p. 954). This impurity might be derived from the arsenical sulphuric acid, in its manufacture. It would be proper to test for arsenic any sample of the sulphate which causes irritation. Arsenic may thus find its way into all medicines in which sulphate of potash is used, *e.g.* Dover's powder.

Chemical analysis.—Sulphate of potassa is easily recognized: it is in the form of hard colorless crystals, soluble in water. Its solution exhibits, with the appropriate tests, the presence of both sulphuric acid and potassa.

As Dr. Taylor justly observes, there is no doubt that the most simple purgative salts may, under certain circumstances, and when given in very large doses, destroy life. Instances have already been cited (*ante*, p. 16) where both sulphate of magnesia (Epsom salt) and chloride of sodium (common salt) caused death, and gave rise to a suspicion of poisoning.

ALUM (*Sulphate of Alumina and Potassa*), in large doses, occasions severe irritation of the stomach and bowels, causing violent vomiting, and sometimes purging. Orfila found it to prove fatal to animals to which he had given it, in the course of a few hours (Ann. d'Hyg., 1842, ii. p. 433). No authentic record exists of death in the human subject, resulting from alum.

The proper *treatment* consists in promoting the evacuation of the stomach by warm diluent drinks, and the administration of hydrate of magnesia.

Chemical analysis.—Alum is a colorless salt, crystallizing in octahedra, and soluble in water; having a sourish, astringent taste; exposed to heat, it fuses and parts with its water of crystallization, being converted into *dried* or *burnt alum.* Its solution will indicate *sulphuric acid* by nitrate of baryta, *potash* by bichloride of platinum, and *alumina* by potash, which precipitates it as a white hydrate, soluble in an excess of the reagent.

CHLORINATED POTASSA AND SODA.—These salts are improperly named *chlorides* of potassa and soda: they are *hypochlorites.* They are frequently known under the name of *bleaching salts*, or *powders*, and are much used, particularly in France, by washerwomen for cleansing clothes. In that country they are commonly known by the name of *l'eau de Javelle.*

The following admirable medico-legal report, by MM. Tardieu and Roussin, of a case of fatal poisoning by chlorinated soda, is abridged from their work, *Sur l'Empoisonnement.* It presents a very satisfactory description of its effects and of the proper method to be pursued for the detection of this poison:

"A child aged six months had been failing in health for some days, without any known cause. Its mother had noticed on several occasions that its lips were strangely white and inflamed, and that its breath had a disagreeable odor, smelling of the eau de Javelle. Coming home one day unexpectedly, she surprised her husband in the act of forcibly giving the child something from a bottle. She seized the child, and likewise the bottle, and ran off to give the alarm. In a few days after, the infant died. On examination, the alimentary canal was found inflamed, though not violently so. Certain experts employed to clear up the case were not able to arrive at any satisfactory conclusion, and the matter was finally submitted by the authorities to us, with instructions to make a full examination, and report upon the following points: (1) Is not the *eau de Javelle*, even when diluted and given in divided doses, a poisonous substance, especially to a child six or seven months old? (2) Might not the gradual absorption of this liquid cause death, without leaving direct traces of the poison? (3) Are not the peculiar odor of this liquid exhaled by the breath, the persistent irritation of the alimentary canal, the vomiting, and the failing health, certain symptoms of poisoning by this liquid?

"The mouth-piece of the vial, submitted for examination, was composed of an alloy of tin and lead. It presented on the interior a whitish spot, slightly adherent, which on analysis proved to consist of the chloride and carbonate of lead. A glass bottle, containing a liquid of a light pinkish color, gave out the distinct odor of hypochlorous acid and chlorine: the analysis of this liquid showed it to be composed exclusively of hypochlorite of soda. It was the opinion of the previous expert, that the liquid swallowed was hypochlorite of potassa.

"The different organs and liquids mentioned below, with the exception of the bottle, gave out no odor of either hypochlorous acid or chlorine: and this could not be otherwise, for reasons given farther on. It should also be remembered that the method used to preserve the organs, by the inconsiderate use of alcohol, was calculated to destroy the last traces of any hypochlorite that might be present. This salt, naturally very unstable, is instantly decomposed, by the mere contact with alcohol, into products that possess none of the characteristic properties of the original.

"The hypochlorites of potash and soda, so much used in the arts, contain a very storehouse of gaseous chlorine, condensed under a solid or liquid form. As soon as these hypochlorites come in contact with the atmosphere, or with an acid, or with organic matters, chlorine is gradually liberated; and, acting according to its natural affinities, it combines with hydrogen, corrodes, disinfects, decolorizes, etc., according to the nature of the substances on which it acts. The uniform and speedy result of this reaction is the complete destruction of the hypochlorite, and the production simply of an inert *chloride*.

"The precise bearing of the above on a medico-legal case is, that after poisoning with a hypochlorite, even though diluted, and although the examination be made immediately after death, not only will there be a failure to detect the poison, but it will be impossible to recover a trace of free chlorine, or of a hypochlorite. . . .

"In the special case before us, the evidence was clear that if the poisoning was occasioned by the *eau de Javelle*, it must have been administered in small doses repeated, since the child was suffering for a considerable time, and died two months after the first symptoms were manifested. As it was also certain that none of the poison could have been taken within six days of death, it was still more impossible, either by the autopsy or by the chemical examination, to recognize the presence of either chlorine or a hypochlorite. The slight traces of the toxic agent had long since been transformed, and, to a great extent, had been eliminated from the economy.

"As to the poisonous effects of these hypochlorites there can be no doubt. If taken in their concentrated state, they may occasion death very rapidly; and even when diluted, and taken in small but repeated doses, their deadly action, although retarded, is not the less certain. The peculiar and characteristic *odor* of the hypochlorites may easily be recognized in the breath of a person who has just swallowed a dose of the compound: this affords a very excellent proof of the presence of the poison.

"From the preceding facts, it follows that the chemical examination for a hypochlorite, several days after death, can yield no positive result, if directed to the detection of free chlorine or hypochlorous acid. For a solution of the problem we must examine the new products resulting from the reaction of the suspected poison, either upon the organs of the body, or upon matters connected with the deceased. In this connection, we derived valuable results from the examination of the urine and kidneys of the deceased child.

"The urine contained a notable quantity of the *chloride of sodium*,—more than half as much again as is usually found in the urine of children of that age. The kidney subjected to complete incineration in a porcelain crucible, likewise furnished an abnormal amount of *chloride of sodium*. The other organs, and the contents of the stomach, contained much smaller quantities. Without wishing to draw any absolute conclusion from these facts, we may remark that the administration of the hypochlorite of soda internally would have precisely the effect of causing an increase of chloride of sodium in the kidneys and the urine,—this being the ultimate product of the decomposition of the hypochlorite.

"The ordinary alloys of lead and tin, so much employed in domestic use, are not sensibly acted upon by neutral liquids, especially by milk. It is quite otherwise, however, when they are in contact with the hypochlorites, even if dilute. The first effect of the contact would be the formation of the *chloride of lead*, in the form of a white deposit. This is precisely what was found on the inside of the mouth-piece seized.

"Finally, the former experts had discovered that one of the little child's caps was stained with numerous spots of a reddish-white color, in the part corresponding to the back of the head. In our opinion, the formation of such stains on a stuff of black woolen, together with their very position, has a significance that deserves the closest attention. In fact, black stuffs generally resist the action of ordinary liquids, and are deprived of their color only by very powerful agents, among which the hypochlorites of commerce occupy the very first rank as decolorizing agents. Any fabric dyed black, if touched with a solution of a hypochlorite, however lightly, will assume a reddish-brown tint. So, also, the discovery of these stains, especially on the back part of the head, may be easily accounted for. Supposing the poison to have been given to the child, it would naturally be in the recumbent posture, and some of the liquid would be very apt to flow (from the instinctive regurgitation and movements of the child), first along the commissure of the lips, then along the ears, until it would finally accumulate at the back of the head, precisely on the borders of the cap, where most of the stains were found. One of the former experts mentioned a superficial inflammation of the cheek and the left ear, for which he could not account. This latter circumstance, taken in connection with the former facts, is not without peculiar significance."

The *conclusion* arrived at was: "That the abnormal proportion of chloride of sodium found in the kidneys and urine of the deceased, the formation of the chloride of lead on the metallic mouth-piece, the presence of numerous stains of a reddish-white color on one of the black woolen caps of the child, the superficial inflammation of its cheek and left ear, the peculiar and characteristic odor of its breath, noticed on various occasions by its mother, and the discovery of a bottle containing hypochlorite of soda, among other articles seized by the officers of the law, constitute, by their precise and logical connection, a series of facts which warrant the conclusion that the case is one of poisoning by hypochlorite of soda.

"There is no doubt that the hypochlorite of either soda or potash, even when diluted with water and administered in small doses, is of a poisonous nature, especially in the case of a child six or seven months old. Moreover, it is certain that the gradual absorption of this substance under the conditions specified might determine a fatal result, without leaving in the organs any material traces of the poison.

"The peculiar characteristic odor of the hypochlorites is one of the most valuable indications of poisoning by this substance. The continued irritation of the intestinal canal, the vomiting, and the wasting of the body are decided symptoms of poisoning by the *eau de Javelle.*"

SALTS OF BARYTA.—The *chloride* and *carbonate* are the two most poisonous salts of baryta. The former has on several occasions been taken by mistake for Epsom salt, and sometimes with fatal effect. The *symptoms* are those of the irritants generally (see *ante*, p. 135), with the addition of violent nervous symptoms, such as cramps, convulsions, headache, excessive debility, dimness of sight and double vision, noises in the ears, and violent palpitation of the heart.

The *morbid appearances* are similar to those produced by the alkalies generally. In one case, in which death took place in two hours, the stomach was found perforated (Guy's Forensic Medicine, p. 417).

Chemical characters.—The chloride of *barium* occurs in irregular tabular crystals, soluble in water; it has an acrid taste. In solution, it is precipitated by sulphuric acid, or a soluble sulphate, as the white sulphate of baryta, insoluble in the strong acids; and by nitrate of silver, as the white chloride of silver, insoluble in nitric acid, but very soluble in ammonia.

Carbonate of baryta, although less powerful as a poison than the chloride, has destroyed life in several cases. The symptoms are similar to those produced by the chloride.

It is a white, insoluble powder, effervescing with acids; completely soluble in dilute hydrochloric acid, from which it crystallizes out by evaporation as the chloride, which may be tested as before mentioned.

The proper *treatment* for poisoning with a barytic salt is the free use of sulphate of soda or magnesia as an antidote; emetics, or the stomach-pump; diluents; and the subsequent use of antiphlogistic remedies.

CHAPTER XIII.

IRRITANTS HAVING REMOTE SPECIFIC PROPERTIES.

This subdivision of Irritant Poisons differs from the Simple Irritants, in adding to the ordinary symptoms of irritation and inflammation of the stomach and bowels common to the former, certain other symptoms which indicate an impression upon the great nervous centres. Several of the alkaline and earthy salts, already described, are of this character, and might have been, very properly, considered under the present head; but they were more conveniently, and perhaps more naturally, discussed in connection with the Alkalies.

SECTION I.

POISONING BY PHOSPHORUS.

Phosphorus is one of the most universally diffused chemical constituents of the animal body: there is not an organ, tissue, or fluid in which it is not found in a state of combination. Its presence is essential to the performance of their normal functions; and this is especially true in relation to the great nervous centres, of which it constitutes a comparatively large proportional part.

Nevertheless, in its *free* state, phosphorus is a most violent poison; and although it is not often employed with homicidal intent, at least in this country (as its odor, taste, and luminosity would be very apt to reveal its presence), several instances have been reported in England where such attempts have been made, but which were happily frustrated through the means just mentioned. Cases of phosphorus-poisoning of an accidental and suicidal character have, of late years, become very frequent, especially in France, where, according to Tardieu, it heads the list of all the poisons, being far more frequently used in that country than even arsenic. This may be accounted for from the facility with which the poison may be procured, in the shape of the common lucifer-matches, and vermin-paste, both of which contain it in considerable quantity.

Symptoms.—Although phosphorus is a violent irritant poison, yet, as a rule, its symptoms do not manifest themselves for some hours after it has been swallowed. The variation in this respect may depend upon the state in which the poison is taken. When, however, they once appear, they are apt to proceed rapidly to a fatal termination. At first a disagreeable, garlicky taste is perceived, which is peculiar to phosphorus. An alliaceous odor may also often be discovered in the breath. There is an acrid, burning sensation in the throat, with intense thirst and nausea; severe pain and heat, with tenderness and pricking sensation of the stomach; followed by distension of the abdomen, and violent vomiting, with occasional purging. There is also cold perspiration, with great anxiety, and small, frequent, and irregular pulse. The matters first vomited generally exhale an alliaceous odor, and are luminous in the dark; their color is very dark green, or like coffee-grounds. The discharges from the bowels have also been observed to be phosphorescent. The pupils are usually dilated, and insensible to light. Sometimes convulsions precede death; at other times the patient dies quietly, in a coma. If the case is protracted for some days, jaundice is apt to occur, and likewise hemorrhage from the stomach, nose, and other parts of the body. The urine is highly albuminous.

Chronic poisoning by phosphorus is accompanied with nauseous eructations, frequent vomiting, a sense of heat in the stomach, purging, pains in the joints, wasting of the body, fever, derangement of the stomach, and diarrhœa, under which the patient slowly sinks. There is a form of slow poisoning produced by inhalation of phosporus-vapor, to which those who manufacture lucifer-matches are exposed. This is a very insidious and fatal form of phosphorus-poisoning. It generally manifests itself first in the jaw, causing caries of the teeth, necrosis of the bone, and abscesses. There is also frequently great irritation of the respiratory organs, together with bronchitis and wasting diarrhœa.

Fatal quantity.—The quantity required to destroy life is very small. Sir R. Christison quotes several cases, one of which proved fatal in twelve days, after taking *a grain and a half;* and another, related by M. Martin-Solon, in which a patient affected with lead-palsy, who took *less than a grain* of phosphorus in the form of an emulsion, was attacked with burning in the gullet and stomach, vomiting, tenderness of the abdomen, general coldness, great prostration, clouded intellect, and death in a little over two days. A child two years and a half old died after swallowing the phosphorus on eight friction-matches; and a child two months old is said to have died from the effects of two such matches (Husemann, Jour. f. Phar., ii. 169). In this case, the quantity was less than one-fiftieth of a grain. Dr. Taylor mentions the case of a lunatic who died from swallowing *one-eighth of a grain* (On Poisons, p. 315).

It has been supposed by some to act fatally in consequence of its conversion into phosphorous acid; but, although such a change may partially occur, it is pretty certain that it is absorbed unaltered into the blood, since the urine voided during life has been observed to be luminous. Hence it is most probably a blood-poison.

Phosphorus is occasionally used in medicine in minute doses—from one-sixtieth to one-thirtieth of a grain, two or three times a day, very gradually increased. Even in these small doses, its effects are uncertain, and it sometimes acts with unexpected severity.

Fatal period. — Phosphorus cannot be classed among the rapidly-fatal poisons. It usually causes death in from one to four or five days; but there is great diversity in this respect. Prof. Casper quotes a case of a young lady who died in *twelve hours* after swallowing three grains of phosphorus in the form of an electuary (Foren. Med., ii. p. 100). Dr. Taylor mentions a case reported by Dr. Habershon, which is said to have proved fatal in *half an hour:* this is the shortest period recorded. Sir R. Christison and other authors speak of the case described by Dr. Flachsland, of Carlsruhe, that of a young man, who took an unknown quantity of phosphorus on bread and butter, at the recommendation of a quack, and in whom death took place in *forty hours*, after intense suffering and continual vomiting, along with a discharge, by the use of injections, of small fragments of phosphorus, which were luminous in the dark, and burnt holes in the bed-linen. The majority of cases of acute poisoning survive for several days. Cases of chronic poisoning may last for several months, or even years.

Treatment.—There is no chemical antidote to this poison. Free emesis should be encouraged, and albuminous and mucilaginous drinks, holding hydrate of magnesia suspended, should be taken largely. The use of oil is objectionable, as this is a solvent for phosphorus, and would consequently tend to diffuse it in the stomach. Of late years, *oil of turpentine* has been recommended as a good antidote (Wharton and Stillé's Med. Jurisp., 1873, vol. ii. p. 349). According to Dr. S. R. Percy (Prize Essay, Trans. Am. Med. Association, 1872), who has experimented extensively with phosphorus upon dogs, the *pure* oil of turpentine (hydrocarbon) is not antidotal; but the *old* oil, which contains oxygen, if administered soon after taking the poison, and before the latter is absorbed, appears to be perfectly so. Dr. Percy found the most reliable antidote to be *oxygenated water* highly charged under pressure, gradually introduced into the stomach by means of an elastic tube. This method, together with inhalations of oxygen, proved remarkably successful in his hands, as an antidote. He also alludes to another antidote, proposed by MM. Eulenberg and Vohl,—*animal charcoal*, in the form of pills: it is said to act antidotally by absorbing the free phosphorus, and thus neutralizing its effects.

Morbid appearances.—According to Tardieu (Sur l'Empoisonnement, p. 437), who has given special attention to this subject, the lesions produced by phosphorus vary according to the form in which it is taken. It is when in the pure state, or simply dissolved in oil, that it most frequently occasions lesions in the œsophagus and alimentary canal. Fragments of phosphorus, recognizable by their odor and phosphorescence, may be discovered adhering to the mucous membrane, even in the large intestines; and at these spots the digestive tube is liable to perforation during the examination. In the œsophagus, stomach, and intestines, ecchymotic or gangrenous spots are scattered about. The mesenteric glands are engorged, and often softened and friable.

When the poisoning has been caused by phosphorus-paste, or by the heads of matches, it may happen that no appreciable lesion is discoverable either in the alimentary canal or elsewhere; but generally, even in the absence of redness, ulceration, or any other evidence of inflammation, many hemorrhagic points can be determined. On opening the abdomen, the mesentery and visceral peritoneum appear studded with black ecchymotic patches or points, resembling the spots of purpura. The pleural and pericardial centres contain more or less of bloody serum. Irregular ecchymotic patches are scattered under the pleura, the pericardium, and even under the endocardium. The heart is soft, discolored, empty, or contains fluid blood; the blood itself is very dark, fluid, and syrupy, but without notable change in its corpuscles. The experiments of Dr. Percy (*loc. cit.*), however, prove that the blood-corpuscles undergo a speedy and complete disintegration, which seems to be the true cause of the numerous ecchymosed spots seen upon the different organs. Bloody infiltrations occur sometimes in the thickness of the viscera, of the muscles, and of the cellular tissue. The bladder contains bloody urine, and often presents submucous ecchymoses.

The exterior of the body often exhibits an icterode hue. As regards the cadaveric rigidity, or the tendency to putrefaction, there is nothing special to notice. It may happen that the red or blue coloring-material used on the tops of the matches (together with small fragments of the wood) will be found sticking to the inside of the digestive canal, even a considerable time after death. M. Dionis (d'Auxerre) was able to detect the presence of vermilion in the remains of a man who was poisoned by soup containing match-heads, *eighteen months* after burial. The general appearance of the gastro-intestinal mucous membrane is hemorrhagic rather than inflammatory,—ecchymoses being scattered over the pyloric region of the stomach, the duodenum, and the large intestine; but without either ulceration or perforation. The contents of the intestines are liquid and bloody.

There is one particular pathological condition of the tissues, revealed by the microscope, that constitutes an important part in the evidence of poisoning by phosphorus: this is a fatty degeneration of the liver, kidneys, glands of the stomach, the heart, and the muscles generally. (See on this subject an inaugural thesis by M. Fabre on "The Fatty Degeneration in Acute Poisoning by Phosphorus," Paris, 1864; also a paper by Dr. Moore, in "Dublin Med. Press," November 15, 1865). M. Tardieu (*loc. cit.*, p. 440) gives an accurate and detailed description of the pathological changes in the different organs, caused by this fatty degeneration; he likewise gives an excellent representation, by magnified drawings, of the microscopic appearances mentioned above.

It should, however, be remembered that these fatty degenerations are not *peculiar* to phosphorus-poisoning, since they occur in poisoning by many other agents, as ammonia, alcohol, arsenic, antimony, the cyanides, and the sulphocyanides; and likewise as the result of certain diseases, acute and chronic. But these changes acquire particular importance from being associated with some of the most notable symptoms of phosphorus-poisoning, such as the jaundice, the muscular pains and weakness, and the albuminous condition of the urine.

The contents of the stomach, in some instances, have evolved the odor and white fumes of phosphorus. In the case of Prof. Casper before mentioned, forty-eight hours after death luminous vapors were observed to issue from the vagina, and a grayish-white vapor, strongly smelling of phosphorus, continuously streamed from the anus! A distinct odor of phosphorus also came from the mouth, but without visible vapor. No smell or vapor of phosphorus could be detected on opening the stomach; nor was any part of its lining membrane either softened or corroded; and no particles of phosphorus could be detected even with the aid of a magnifying-glass. It contained about half a pint of a bloody fluid, mingled with coagulated milk. The intestines were pale, and presented nothing abnormal. The blood was dirty red, and of a syrupy consistence; and the blood-corpuscles were transparent and deprived of their coloring-matter. The liver, spleen, and kidneys were congested; the lungs contained but little blood; and the heart was almost completely empty; but the large blood-vessels contained much blood. The bladder was of a livid color, and contained about a tablespoonful of milky urine. The brain and likewise its membranes were moderately congested.

In Dr. Flachsland's case, referred to on page 196, which proved fatal in forty-eight hours, watery blood flowed in large quantity from the nostrils, and also from incisions made into the skin and muscles of the abdomen; the stomach and bowels externally were inflamed; the mucous membrane of the stomach presented a gangrenous inflammation, which extended into the duodenum; the large intestines were contracted to the size of the little finger. The mesenteric glands were hardened; and the spleen and kidneys inflamed.

In other cases, the lining membrane of the stomach has been found of a crimson color, softened in many places, and easily detached, with ulcerations in different portions of the organ; the small intestines violently inflamed, together with the rectum. On the other hand, in several instances, where very large doses were taken, the post-mortem examination failed to reveal the slightest lesion of the stomach, or indeed of any of the organs, visible to the naked eye: doubtless, however, if the microscope had been employed, the fatty degeneration of the different tissues above described, would have been detected.

Diagnosis.—During life, the symptoms of a case of acute phosphorus-poisoning are commonly sufficiently distinct to admit of an easy recognition. If in addition to the gastro-enteric symptoms already detailed (*ante*, p. 194), a phosphorescent vapor is perceived to issue from the mouth, accompanied with a garlicky odor; and if, further, a similar phosphorescence is observed in the discharges from the stomach and bowels and the urinary bladder, there can be no difficulty in arriving at a positive conclusion. In chronic cases accompanied by jaundice, it is possible that the case might be confounded with the disease known as *grave* or *pernicious icterus*, *acute yellow atrophy of the liver*, or *general spontaneous steatosis.* M. Tardieu (*loc. cit.*, p. 444) is of the opinion that many reputed cases of this disease are in reality only concealed cases of poisoning by phosphorus. This, moreover, appears to be the opinion of the majority of the German authorities, notwithstanding the contrary idea of Wunderlich. Tardieu cites the following points of distinction between the two disorders. As a general rule, the primary symptoms of phosphorus-poisoning are less severe than those of spontaneous icterus; the sensation of heat in the throat, and eructations, and vomiting of matters of a garlicky odor and which are luminous in the dark, would indicate the former. The yellow discoloration of the skin shows itself at a later period in the poisoning than in the disease: it is not so intense, nor is it accompanied with the injection of the eyes, or with the fever, which are never wanting in acute icterus. Moreover, the disease lacks the intermissions and the periods of prolonged sedation which are observed in cases of poisoning.

As regards the fatty degeneration of the liver and other organs, it would seem, from well-instituted experiments, that this pathological change is brought about more speedily in phosphorus-poisoning. The experiments of MM. Fritz, Ranvier, and Verliac on animals that were made to take phosphorus, proved that a steatosis of the liver had been produced in a few days; and Tardieu speaks of having himself witnessed this fatty change in the heart, liver, kidneys, glands of the stomach, and the muscles generally, in forty-eight hours after the ingestion of this poison (*loc. cit.*, p. 445).

Chemical analysis.—Phosphorus is a white, transparent solid, of the appearance and consistency of wax; sp. gr. 1.83. It fuses at the temperature of 110° F.; at a higher temperature it takes fire, burning with a brilliant white light, becoming converted into the white fumes of anhydrous phosphoric acid. At ordinary temperatures, when exposed to the air, it gives off white fumes of phosphorous acid, which are faintly luminous in the dark. The smell and taste of phosphorus resemble those of garlic; by which means it may easily be recognized when mixed with food or drinks. The fuming of phosphorus in the air, as also its luminosity, is completely prevented by the presence of the vapor of alcohol, ether, oil of turpentine, chloroform, and ammonia, even in minute quantities.

Although insoluble in water, phosphorus may impart poisonous properties to this liquid from the production of phosphorous acid. It is tolerably soluble (especially by the aid of heat) in the fixed and volatile oils; also in ether, chloroform, and naphtha; but its best solvent is the bisulphide of carbon. It is insoluble in hydrochloric acid; hot nitric acid converts it into phosphoric acid.

Phosphorus is always preserved under water, to prevent oxidizement: after exposure thus for some time to the light, it loses its translucency, and becomes covered with a whitish film, which is believed by Rose to be merely another molecular form of the substance.

Phosphorus in the *free state* is so readily identified by its physical properties—especially by its odor, luminosity, and incandescence—that chemical proofs need hardly be required. These, however, are readily furnished. If a minute portion of free phosphorus is put into a flask containing the materials for generating hydrogen, *phosphuretted hydrogen gas* is evolved, which is luminous in the dark, and is sometimes spontaneously inflammable. If this gas be ignited at the extremity of a drawn-out tube, it will burn with a greenish-blue flame. A paper moistened with nitrate of silver exposed to the unignited gas is immediately blackened. When the gas is made to pass through a solution of nitrate of silver, the latter is blackened from the production of metallic silver: phosphoric acid is also formed in the solution, and can be detected on filtration, by the appropriate reagents. When conducted into a solution of corrosive sublimate, the gas produces a yellowish precipitate.

The methods of Mitscherlich and Lipowitz, which are peculiarly appropriate to the detection of the poison in organic mixtures, will be described in the succeeding paragraph.

Detection in organic mixtures, and in the contents of the stomach.—Very often a strong suspicion, if not the certainty, of the presence of phosphorus in such mixtures is produced by the whitish fumes exhaled, which have a garlicky smell, and are luminous in the dark. If the mixture to be examined is ammoniacal from putrefaction, it must first be acidulated with sulphuric acid, before it is examined for its luminosity, since the presence of free ammonia prevents this display. It is quite possible, however, that the peculiar odor may be disguised, or overpowered, by others of a more powerful kind. All organic mixtures should be carefully searched for particles of the poison, which, if found, may be washed in water and alcohol, and preserved for future examination. Or, the mass may be spread out over a metallic plate, and warmed; on stirring it, the presence of phosphorus will be made evident by frequent bright scintillations. Or, after collecting the suspected particles and washing them, they may be gently heated in a glass tube or flask with water, when they will melt and collect into a globule, which will solidify on cooling, and may easily be identified.

Owing to its great solubility in bisulphide of carbon, phosphorus may be separated from many organic matters by digestion with this liquid. In this way it can be procured from flour and phosphorus-paste, or from the residue of the contents of the stomach, after washing and decantation. On the spontaneous evaporation of the sulphide, decanted from the organic liquid or solid, the phosphorus may be procured in small globules or beads, which will ignite on being touched with a hot wire, and burn with the characteristic bright flame.

If, however, the phosphorus be in a state of solution, or be present in too small a quantity to be dissolved out by sulphide of carbon, its presence may be indicated by one of the following methods:

Process of Mitscherlich.—This is the most delicate method yet devised for the detection of phosphorus. It consists essentially in distilling the suspected mixture, acidulated with sulphuric acid, and condensing the vapors in a glass tube surrounded by a condenser (like a Liebig's condenser): the vapor of phosphorus thus condensed produces a continuous luminosity, easily visible in the dark. The suspected material, diluted with water, if necessary, and acidulated with sulphuric acid, is put into a glass flask, which is connected by means of a bent glass tube with another tube that passes into the glass condenser. The latter is kept surrounded with cold water, in the usual manner. On gently heating the flask, a very distinct and continuous luminosity, usually some inches in length, is observed, in the dark, to play up and down in the cooled portion of the delivery-tube. The phosphorus thus distilled collects with the aqueous vapor in the receiver, to which it imparts the usual alliaceous odor. A portion of it may even collect in the receiver in the form of small globules or beads, which may be easily recognized.

So delicate is this process by distillation that, according to Dr. Taylor, in an experiment with the head of one lucifer-match, the luminosity appeared for half an hour in the condensing-tube. The most absolute darkness is required for the success of this experiment. Prof. Wormley states (Micro-Chem. of Poisons, p. 202) that he distilled the *fiftieth* part of a grain of phosphorus along with two thousand grains of water acidulated with sulphuric acid. A phosphorescent light appeared in the cooled tube, several inches in length, and continued for thirty-four minutes. The distillate had a strong alliaceous odor, and furnished evidence of the presence of phosphorous acid, although it contained no globules. The amount of phosphorus that passed through the tube per second must have been something less than the one-hundred-thousandth of a grain: yet this gave a luminosity many times greater than would have sufficed to recognize its presence with absolute certainty. We have ourselves verified this experiment with a granule containing one-sixtieth of a grain. It should not be forgotten that the presence of alcohol, chloroform, etc. (substances in which the viscera of a body may have been preserved), may entirely prevent the luminosity in the above experiment. But as the more volatile bodies are gradually dissipated by the heat, their interference ceases, and the luminosity will become evident.

The presence of phosphorus in the distillate (in the form of phosphorous acid) is best shown by first treating the filtered liquid with a little nitric acid, which converts it into phosphoric acid; then concentrating, and applying the appropriate tests (see *post*). This examination of the distillate will be unnecessary if globules of phosphorus have been detected in the receiver. On the other hand, if no globules or luminosity have been observed, the discovery of a small quantity of the oxides of phosphorus in the distillate is not sufficient to warrant the supposition of poison, since these might have been carried over mechanically. It is to be remembered that it is only *free* phosphorus that gives out the luminosity by the above process. The distillation of the blood, brain, or other protein-compounds of animals (which contain it in the state of combination), along with dilute sulphuric acid, entirely fails to produce it.

Method of Lipowitz.—This consists in boiling the suspected liquid, slightly acidulated with sulphuric acid, with fragments of sulphur, in a retort with a long neck, or one which may be connected with a cooled glass tube (as in the method of Mitscherlich), the experiment being conducted in the dark. The sulphur has the property of abstracting the phosphorus from even complex mixtures, and combining with it. The boiling is continued for about half an hour, after which the pieces of sulphur are withdrawn and washed with water. They will now emit the peculiar odor of phosphorus, and be luminous in the dark. On gently heating them with nitric acid, they yield a solution containing phosphoric acid, together with sulphuric acid. On evaporating this to a small volume, diluting and filtering, the presence of *phosphoric acid* may be recognized by the usual tests.

As the distillation proceeds, the peculiar luminosity will usually be perceived in the condenser; and the distillate will also reveal the presence of the oxides of phosphorus. If, however, the amount of phosphorus present in the original mixture was very minute, the whole of it may be retained by the sulphur.

The hydrogen method.—This process depends upon the property possessed by free phosphorus and its lower oxides, of uniting with nascent hydrogen to form phosphuretted hydrogen, a gas easily recognized by its peculiar properties. The suspected material, properly prepared, should be introduced into the flask containing the materials for generating hydrogen; the purity of the latter gas is first proved, before the phosphorus mixture is introduced. The resulting phosphuretted hydrogen is made to pass over hydrate of potassa or lime, in order to remove any sulphuretted hydrogen that might be present; it is then ignited as it escapes from the drawn-out end of the delivery-tube, when it will burn with a characteristic greenish flame, which disappears when the tube becomes heated. If a piece of cold porcelain be depressed upon the flame, the latter burns with an emerald-green color at the point of contact, until the porcelain becomes heated. Moreover, the evolved gas has a peculiar odor, and is luminous in the dark; it likewise yields a black precipitate with the nitrate of silver. Fresenius states that by this method the presence of phosphorus can be recognized, even when in very small quantity and mixed with putrid animal matters; and also in the presence of substances which interfere with the luminosity by the process of Mitscherlich.

Failure to detect the poison.—It may readily happen that, owing to the length of time that has elapsed since the phosphorus was swallowed, it has either all been eliminated from the system, or what remains has been oxidized into phosphorous, or even phosphoric, acid. In such a case it can no longer be detected, either by the method of Mitscherlich or of Lipowitz. If it exists in the form of *phosphorous acid*, the hydrogen method will still serve to discover it; but if it has been oxidized to *phosphoric acid*, other processes must be resorted to, which will now be detailed.

The suspected mixture, after dilution (if necessary), and filtration, is treated with a small quantity of nitric acid, and concentrated by evaporation: this serves to convert all the lower phosphorous oxides into phosphoric acid. It is then treated with a slight excess of pure carbonate of soda, evaporated to dryness, and fused in a porcelain crucible. By this process all the organic matter is destroyed, and pure tribasic phosphate of soda remains. This is dissolved in water, and the usual tests applied.

This method of testing for phosphorus in a suspected case of poisoning is by no means satisfactory, since phosphoric acid in combination with the alkalies and earths is found in the different animal tissues, and also in many articles of vegetable and animal food.

The exact time when failure to detect phosphorus after its administration occurs, is not definitively settled. It must evidently depend on the usual contingencies of early and copious vomiting, early treatment, etc. One instance is recorded where it was discovered in the contents of the stomach and bowels on the *tenth* day after it was taken; another, where it was found in the free state in the stomach after *fourteen* days' burial, and where, moreover, there was considerable decomposition. The longest period recorded is in the case of a child three and a half years old, poisoned by phosphorus-paste. A grain of phosphorus was obtained from the stomach and intestines after the body had been buried *three weeks* (Dr. Ludwig, Jour. de Chim. Méd., 1863, p. 584).

Quantitative determination:—Any phosphorus obtained in the solid state, after washing and drying, may be carefully weighed as such. If, however, it has been converted into phosphoric acid, it is determined as *pyrophosphate of magnesia.* For this purpose, the solution is treated with sulphate of magnesia, ammonia, and chloride of ammonium, and allowed to stand for several hours, to permit the complete separation of the precipitate, which is ammonio-magnesian phosphate. This is collected on a filter, washed with water containing a little ammonia, dried, ignited (which drives off the ammonia), and, after cooling, weighed. Every 100 parts of the pure ignited residue correspond to 64 parts of anhydrous phosphoric acid, or 28 parts of phosphorus.

PHOSPHORIC ACID.—This acid, together with *phosphorous* acid, has proved fatal to the lower animals, when administered to them in large quantities. It has been supposed by some that the fatal effects of phosphorus on the human subject were owing to its conversion into these compounds. This opinion, however, cannot be maintained, as it is opposed to experience.

The chemical tests for *phosphoric acid* are the following:

1. *Nitrate of silver.*—Free phosphoric acid is not precipitated by this reagent; but from neutral solutions of the alkaline phosphates it throws down a light-yellow *tribasic phosphate of silver*, which is readily soluble in ammonia, and in nitric, acetic, and free phosphoric acids. Hydrochloric acid converts it into the white chloride. Nitrate of silver also precipitates a neutral solution of arsenious acid, of a light-yellow color, soluble in ammonia and in free acids; but the precipitates are easily distinguished from each other by drying, and heating them in a reduction-tube: the latter yields a sublimate of octahedral crystals of arsenious acid, while the former is not at all affected. Again, if the arsenious solution be acidified with hydrochloric acid and boiled with a piece of bright copper, it will produce a deposit of metallic arsenic; no such effect is caused by the phosphatic solution.

Nitrate of silver also yields yellowish precipitates with the iodides and bromides; but these are insoluble in dilute nitric acid, and only sparingly soluble in ammonia.

2. *Sulphate of magnesia and ammonia.*—A mixture of sulphate of magnesia, ammonia, and chloride of ammonium produces with phosphoric acid, both free and in combination with alkalies, a characteristic crystalline precipitate—the *ammonio-phosphate of magnesia.* The crystals have a beautiful feathery stellate appearance, and may be distinctly identified under the microscope from a solution as dilute as the one-hundred-thousandth of a grain in a drop of water.

Arsenic acid also produces with the ammonio-sulphate of magnesia a similar crystalline precipitate: the latter, however, when dissolved in just sufficient acetic acid, will yield a reddish-brown precipitate with nitrate of silver; while the phosphatic precipitate treated in the same way, yields a *white* precipitate.

3. *Molybdate of ammonia.*—This reagent should first be mixed with sufficient nitric or hydrochloric acid to redissolve any precipitate that forms. A small quantity of this mixture is put into a test-tube, and a few drops of the phosphoric solution are added, when the mixture will acquire a yellow color, and the yellow pulverulent precipitate of *phospho-molybdate of ammonia* will be thrown down. A gentle heat greatly promotes this reaction. It is requisite that the molybdate of ammonia should be in very strong solution. This precipitate is insoluble in the strong acids, even on boiling; but it is readily soluble in excess of free phosphoric acid, in the alkaline phosphates, the caustic alkalies and their carbonates, and the alkaline tartrates.

RED, AMORPHOUS, OR ALLOTROPIC PHOSPHORUS.—This remarkable variety of phosphorus is prepared by exposing ordinary phosphorus to a heat of 450° F., in an atmosphere deprived of oxygen (as in carbonic acid gas), for a number of hours, when it will be found changed into a hard, dark-red, brick-like mass, from which any unchanged phosphorus may be dissolved out by means of bisulphide of carbon.

Red phosphorus, although identical with the common variety in its chemical composition, is totally distinct from it in physical, chemical, and physiological properties. The differences between the two are clearly shown by a reference to the following table, taken from Dr. Percy's essay (*loc. cit.*):

Common Phosphorus.	*Red Phosphorus.*
Poisonous.	Innocuous.
Evolves a strong odor.	Nearly odorless.
Phosphorescent, luminous in the dark.	Not phosphorescent.
Melts at 108° F.	Melts at about 500° F.
Transparent.	Opaque.
Almost colorless.	Varies in color from reddish-black lustrous, to iron-gray, brick-red, crimson, and scarlet.
Freely soluble in various liquids.	Nearly insoluble in all liquids.
Distinctly crystalline.	Amorphous.
Soft; may be indented with the nail.	Hard as red brick.
Flexible as copper or lead.	Brittle as glass.
Oxidizes in the air at ordinary temperatures.	Unalterable in the air.
Unites readily with other elements.	Is acted on by other elements with great difficulty.

To the above points of distinction we may add that nitric acid acts upon common phosphorus with great energy, oxidizing it to phosphoric acid: it produces no effect on the amorphous variety; and while the former combines with chlorine gas with active combustion and flame, the latter combines with it without flame.

When the red, amorphous variety is heated to the boiling-point in a gas devoid of oxygen, it is again restored to the state of ordinary phosphorus. Red phosphorus is now very extensively used, especially in Germany, in the manufacture of matches. It is hoped that this may lead to the abandonment of the poisonous variety for this purpose.

SECTION II.

POISONING BY IODINE AND BROMINE.

Iodine occurs in brilliant dark-colored scales, resembling coarse iron-filings; it has a peculiar disagreeable odor; and gives off, when heated, violet fumes, which are powerfully irritating to the nostrils, throat, and lungs. It is very sparingly soluble in water, but readily so in alcohol and ether, and also in the aqueous solution of iodide of potassium. It is found in the shops in substance, and in the forms of *tincture* and *compound tincture.*

Symptoms.—Like phosphorus, iodine produces both a local and a remote effect on the system. The latter is the usual result of the continued use of iodine in small doses. In large doses it acts as a powerful irritant, causing burning heat in the throat, severe pain in the abdomen, with vomiting and purging, the vomited matters having the peculiar odor of iodine, and being of a yellowish color, except when farinaceous food had been taken, in which case they are blue; sometimes they are mixed with blood. The discharges from the bowels may also contain iodine, if it has been administered in the solid state. Besides these symptoms, there are others indicating the absorption of the poison, and its influence upon the nervous system, such as giddiness, headache, thirst, with anxiety, trembling, convulsive movements of the limbs, and fainting.

In chronic poisoning (*iodism*) produced by the prolonged employment of iodine, in medicinal doses, or by its external application, the symptoms are vomiting and purging, tremors, palpitation, pain in the stomach, cramps, salivation, general emaciation, and gradual absorption of certain glands of the body, particularly the mammæ of females and the testes of males: there is usually an increase of most of the secretions, and enlargement and tenderness of the liver. All of these symptoms are not generally present in every case of iodism, but they have been produced by small doses administered for a few days at a time.

There is a singular uncertainty in the action of large doses of iodine, some persons appearing to be insensible to its impression. Sir R. Christison (On Poisons, p. 194, *et seq.*) mentions cases illustrating this. Dr. Kennedy gave an average of twelve grains daily, in the form of tincture, for eighty days, without observing any effect at all; Mr. Delisser gave a patient thirty grains in one day, without injury (quoted in Dr. Cogswell's Experimental Essay, p. 23); and Dr. S. Wright met with the case of an infant three years old, who took three drachms of the tincture at a dose, and only suffered from attempts to cough, some retching, and much thirst (*ibid.*, p. 27). On the other hand, severe symptoms have been observed to commence when half a grain was taken three times a day, for a single week (Gairdner, On the Effects of Iodine, p. 20). Even small medicinal doses, frequently repeated, have been known to break out, like digitalis, mercury, and other poisons, with alarming violence, the symptoms being those of excessive irritation.

A fatal case is related by M. Zink, a Swiss physician. His patient, after taking too large doses of iodine for about a month, was seized with restlessness, burning heat of the skin, tremors, palpitation, syncope, excessive thirst, very frequent pulse, violent priapism, and copious diarrhœa of bilious and black stools. Death occurred after six weeks' illness. Another case is related in the Provincial Journal, June, 1847, p. 356, in which one drachm of the tincture in about an ounce of spirit is said to have proved fatal.

Morbid appearances.—Redness of the mucous membrane of the stomach and bowels, in some places approaching to gangrenous discoloration; corrosion of the lining membrane of the stomach, which may be detached in large patches; enlargement of the liver; contraction and redness of the œsophagus; adhesion of the abdominal viscera to one another, with serous effusion into the peritoneum, and sometimes into the pleura.

Chemical analysis.—In the solid form, iodine is easily recognized by its physical properties, above mentioned. When its solution is added to cold boiled starch, it instantly imparts to it a fine blue color, which disappears on boiling, and reappears on suddenly cooling. The color disappears permanently by a stream of sulphuretted hydrogen. This is a delicate and characteristic test. If it exist in combination, as an iodide, the iodine must first be liberated before the starch test will act: this is best accomplished by adding fresh chlorine-water, which decomposes the iodide; an *excess* of chlorine will bleach the blue color, and cause it to disappear. The mineral acids will also liberate iodine from the iodides.

In organic mixtures.—Iodine may be recovered from organic mixtures, if in the free state, by the action of bisulphide of carbon, which freely dissolves it, forming a rich pink solution. By decanting the watery liquid from the sulphide solution, and allowing the latter to evaporate, the iodine will be left in the form of crystals.

As many organic substances convert iodine into hydriodic acid, it is usually to be sought in the latter combination. After dilution, if necessary, and filtration, the starch test may be applied, provided the color of the solution is not too deep. If this is the case, it is recommended to agitate it with one-third its volume of ether, which will separate the free iodine, and will yield the appropriate reaction with starch. Should this process fail, the iodine has probably been converted into an iodide. In this case, Sir R. Christison (On Poisons, p. 199) advises the following course: Add water if necessary, and filter: if the filtrate is tolerably free from color, test a little of it with solution of starch and chlorine. If the color is too deep to admit of this trial, or if the test fails, unite the fluid and solid parts, and transmit sulphuretted hydrogen, to convert any free iodine into hydriodic acid. Drive off the excess of gas; supersaturate with a considerable excess of potassa, filter, and evaporate to dryness. Char the residue at a low red heat in a porcelain crucible; pulverize the carbonaceous mass, and exhaust with water. Evaporate to dryness, and again exhaust with alcohol, which takes up the iodide of potassium and some other salts, leaving some of the salts behind. On evaporating the alcoholic solution to dryness, a residuum is left, which, when dissolved in water and filtered, will give the characteristic reaction with starch and chlorine.

By the above method, one grain of iodide of potassium may be detected in six ounces of urine.

Iodide of potassium, although very much employed in medicine, in doses as large as five to thirty grains several times a day, occasionally produces violent effects on the system, such as griping pains in the abdomen, headache, thirst, frequent pulse, dyspnœa, and inflammation of the nostrils and eyes. A pustular eruption also often attends its use, and salivation is an occasional symptom. As found in the shops, this salt is often greatly adulterated, containing sometimes as much as fifty to nearly eighty per cent. of carbonate of potash. (Pereira, Mat. Med., i. p. 489.)

Iodide of potassium may be detected in the blood, liver, spleen, muscles, and especially in the urine.

Bromine.—This is a dark-red, volatile liquid, possessing a very pungent, unpleasant odor, and an acrid taste. The vapor is exceedingly injurious to the eyes and lungs. It is a highly corrosive fluid, acting upon and destroying animal tissues with great rapidity. As bromine is much employed in daguerreotyping, it may readily become the cause of poisoning. The only case of fatal result is reported by Dr. Sayre, of New York. A daguerreotypist, aged twenty-four years, of good health and temperate habits, swallowed *one ounce* of bromine, for the purpose of self-destruction. The immediate symptoms were spasmodic action of the muscles of the larynx and pharynx, and great difficulty of respiration. This was soon followed by intense burning heat in the stomach, with great anxiety, restlessness, and trembling of the hands. The pulse was rapid, tense, and corded, and the breathing greatly hurried. The stomach was entirely empty at the time of taking the poison. Various means were tried, unsuccessfully, for his relief, and the symptoms above described increased in intensity; the hands and feet became cold, with failure of the pulse, etc., and death took place *seven and a half hours* after swallowing the poison.

On examination, the external surface of the stomach was found vividly injected, as was also the peritoneal coat of the duodenum and the mesentery. The viscera lying near the stomach were stained of a deep yellow color. A softened ecchymosed spot, one inch and a half in diameter, and several others of smaller size, were found upon the peritoneal coat of the stomach. This organ contained about four ounces of thick fluid resembling port-wine dregs, and exhaling a faint odor of bromine. Its whole internal surface was covered with a thick black layer, resembling coarse tanned leather. The mucous membrane was very thin, and there was intense submucous injection. (Wharton and Stillé's Med. Jurisp., from New York Jour. of Med., Nov. 1850.)

Chemical analysis.—Bromine may be separated from organic mixtures either by bisulphide of carbon or by ether, after the method described for iodine (*ante*, p. 211). If it exists in the form of a *bromide*, this may be decomposed by means of chlorine, or by one of the mineral acids. Bromine is characterized by imparting a deep *yellow* color to boiled starch.

Chlorine is a powerfully irritating gas, of a greenish-yellow color. If inhaled into the lungs, it may destroy life by its irritating effects. Orfila found that a strong solution of chlorine given to dogs, caused death in from one to four days; the symptoms being those of a powerful irritant to the stomach.

Chlorine is readily recognized by its color and odor, and especially by its powerful *bleaching* properties.

CHAPTER XIV.

POISONING BY ARSENIC (WHITE OXIDE OF ARSENIC—ARSENIOUS ACID).

The term *Arsenic* is always employed in Toxicology (unless otherwise qualified) to signify the white oxide of arsenic, or arsenious acid. The *metal* itself is very rarely used for poisonous purposes, although in the form of *fly-powder*—a mixture sold in the shops, consisting of metallic arsenic and arsenious acid—it has frequently occasioned death, but chiefly as the result of accident. The symptoms and morbid changes produced by this substance are essentially the same as those occasioned by arsenious acid.

Arsenic is by far the most important of all the metallic poisons, whether considered as to the frequency of its use, the facility of procuring it, the ease of administration, or the extent of its employment, both in medicine and in the arts. It enters extensively into the composition of the earth, being found in the form of the metal, arsenious acid, the two sulphides (realgar and orpiment), and as a constituent of several ores of iron, copper, silver, tin, zinc, nickel, and cobalt. Most of the arsenious acid of commerce is prepared from a native arsenical sulphuret of iron, known as *mispickel.*

Much of the sulphuric acid found in the shops is contaminated with arsenic, derived from the iron pyrites used in its manufacture; and as this acid, in its turn, is employed in making nitric and hydrochloric acids, and other chemical products, the impurity is carried into these likewise. The two metals zinc and copper, especially the former, are often found to contain arsenic.

In the arts, arsenious acid enters into numerous compounds, as in the manufacture of glass and of white enamel. Composition candles sometimes contain it. It is employed to prevent "furring" in steam-boilers; ship-builders mix it with tar to protect timber from worms; it is sold in powders for destroying rats and other vermin; farmers use it to preserve grain for seed, also as an ingredient in dipping compounds for sheep. Grooms give it to their horses to improve their coats; and there seems to be sufficient testimony to warrant the belief that the peasants of Styria, as also the inhabitants of other countries, habitually take arsenic for the purpose of improving their complexions and rendering themselves capable of greater physical endurance. (See Dr. Roscoe's paper to the Manchester Philos. Society, Oct. 30, 1860; also, Dr. Maclagan's paper in Edin. Med. Jour., vol. x. p. 200; also, letter from Dr. Knapp, *ibid.*, p. 609.)

Besides arsenious acid, the other preparations of arsenic which are important in a medico-legal view are the *yellow sulphide* (*orpiment*), the *arsenite and aceto-arsenites of copper* (*Scheele's green and Paris green*), and the *solution of arsenite of potash* (*Fowler's solution*).

Metallic arsenic sublimes at 356° F. (Prof. Guy has found that small quantities sublime at 230°.) Its vapor has the odor of garlic, similar to that of the vapor of phosphorus. Heated in close vessels, it is deposited unchanged; but heated with access of air, it is oxidized, and deposited in brilliant octahedral crystals.

Properties of arsenious acid.—It occurs in commerce in the form of masses, or as a white, heavy powder. The cake when first sublimed is translucent, but on exposure to the air it becomes opaque, resembling white enamel. It is nearly tasteless, or has only a faint sourish taste. It is erroneously described as being acrid to the taste. Its comparative want of taste renders it easy to be administered with a criminal intent, or to be swallowed by mistake. Its solubility in water depends on the manner in which it is treated: thus, water boiled for an hour on arsenious acid, and allowed to cool, dissolves the fortieth part of its weight, or about twelve grains to the ounce. If boiled for a shorter time, not more than one-eightieth part will be dissolved; cold water allowed to stand for many hours on the poison, does not dissolve more than the one-thousandth to one five-hundredth part of its weight, or about half a grain to one fluidounce of water. (*Vide* Taylor's Med. Jurisp., Am. ed., 1873, p. 195.) Its solubility is much increased by the addition of an alkali, but diminished by the presence of organic matter. It is very soluble in ammonia, hydrochloric acid, and carbonate of potassa. Its solution deposits octahedral crystals on evaporation. It possesses a slight acid reaction, combining with alkalies, and forming soluble *arsenites.*

When arsenious acid is heated to the temperature of 370° to 400° F. (about 280°, according to Prof. Guy, Forens. Med., p. 429), it sublimes in the form of a white vapor, which is inodorous, and is deposited on a cool surface either as an amorphous powder or in octahedral crystals. If it be thrown upon red-hot charcoal, the odor of the vapor will be alliaceous, because the carbon has reduced the arsenious acid to the metallic form, the vapor of which, as already stated, has the garlicky odor.

Symptoms.—These usually set in within an hour after swallowing a poisonous dose. There is generally first a sense of faintness, attended with a feeling of heat and constriction of the throat, and with thirst, nausea, and burning pain in the stomach. The pain soon becomes excruciating, and is attended with violent retching and vomiting. The matters vomited are sometimes streaked with blood; the pain in the stomach is increased by pressure, and soon extends over the whole abdomen. There is generally severe purging, with tenesmus, and frequently bloody discharges from the bowels. The thirst is usually intense; and sometimes there is great difficulty in swallowing. The features are collapsed, and expressive of great pain; the pulse is small, quick, and irregular; the skin cold and clammy, but occasionally hot; the eyes red; the tongue dry and furred; the respiration labored. Sometimes there are violent cramps in the legs and arms. The urine is often partially suppressed, and passed with pain. In certain cases, stupor, delirium, and convulsions are manifested. In many instances death takes place calmly, the intellect continuing undisturbed to the last. When the poison proves rapidly fatal, death commonly takes place by collapse, or by coma. In more chronic cases, the patient dies exhausted by the violence of the irritative fever, or after a long train of nervous symptoms, terminated by convulsions. Patients who recover may suffer for a length of time from indigestion, from a paralysis of the limbs, or from epileptic fits.

It must be remembered, however, that cases of poisoning by arsenic present a very great variety of character, combination, and severity of the symptoms: consequently we should be prepared for exceptional and anomalous cases. Thus, in one class of cases the symptoms are those above described, in an aggravated form, indicating excessive irritation of all the mucous membranes, along with intense headache and giddiness, incessant restlessness, violent cramps, followed, if life be prolonged, by convulsions, tetanic spasms, epileptic fits, delirium, and coma—symptoms indicating excessive nervous disturbance. In another class the symptoms are those of collapse. There is little or no pain, vomiting, or diarrhœa; but a cold and clammy skin, extreme prostration, very frequent and feeble pulse, or else one very slow and weak; the mind is very slightly impaired, but there is some approach to coma, slight cramps and convulsions, and death, without reaction, in four or five hours. Christison reports a case of this kind, where death took place in four hours, without any vomiting, although the patient had taken emetics. Mr. Fox reports a similar case (Lancet, Nov. 4, 1848), in which a stout, healthy young man took a teaspoonful of arsenious acid by mistake. No marked symptom was exhibited for nearly six hours, when purging suddenly supervened, and he vomited two or three times; he then became drowsy; his countenance sunken and livid; pulse rapid and extremely feeble; body cold and clammy; involuntary stools of a watery character; no pain or tenderness of abdomen; no tenesmus; mind rational. Soon afterwards he complained of dimness of sight, lay down on the bed, and in a few minutes expired.

In a third class of cases the patient falls into a profound sleep, deepening into coma, and dies in a few hours, without rallying. Such a case is reported by Mr. T. Wright, of Dublin (Lancet, vol. xii. p. 194), where death took place in four hours, succeeding coma. The autopsy here revealed no trace of inflammation of the mucous membrane of the stomach, even in the spots covered with the arsenic.

In a fourth class the symptoms very closely resemble those of cholera, so much so as easily to be confounded with them. Such was the case of the Duke of Praslin, who died in Paris in 1847, from arsenical poisoning.

In most cases of acute poisoning by this substance the symptoms steadily run their course; yet sometimes there is a marked remission, or even an intermission, of the most prominent symptoms, lasting for several hours; after which these return with increased violence. In some instances this remission has been repeated several times in the same case.

All the above varieties occur under large and small doses; and they cannot be accounted for by the quantity, the form, or the mode of administration of the poison. Among the occasional symptoms, a livid appearance of the face, jaundice, and an eczematous eruption have been noticed. It has often happened that in a case of fatal poisoning by a very large dose of arsenic, and where the stomach was found intensely inflamed, the patient had complained of no previous pain whatever. Arsenic would thus appear, in some instances, to destroy sensibility.

The symptoms of *chronic poisoning* usually result from frequent repetition of a dose of the substance which of itself is too small to be followed by fatal effects. The most prominent symptoms are great distress about the stomach, with the ordinary signs of indigestion; a sense of burning experienced throughout the alimentary canal; frequent nausea and vomiting, and painful diarrhœa; inflammation of the eyes; intolerance of light; headaches; giddiness; a jaundiced skin; cutaneous eruptions; local paralysis; exfoliation of the cuticle and of the skin of the tongue; falling out of the hair; salivation; great emaciation; hemorrhage of the nose; petechiæ; pain of the joints and back; and a general wearing out of the whole system.

The time when the symptoms first manifest themselves varies considerably. In most instances they appear within half an hour, or an hour, after taking the poison. Cases are reported where the symptoms appeared in the act of swallowing it; others, where they were manifested in eight, ten, and fifteen minutes after. On the other hand, numerous instances are recorded where the time was protracted for many hours after taking it. The longest interval observed was in a case mentioned by Dr. Wood (U. S. Dispensatory, 1865, p. 26), where a drachm had been swallowed, and the symptoms of poisoning were delayed for *sixteen hours.* The great discrepancy in regard to the period when the symptoms manifest themselves may be explained in part, at least, by the condition of the stomach at the time of receiving the poison. If it were swallowed soon after a meal, while this organ was full, absorption would necessarily be retarded, and the operation of the poison would be delayed. Again, if swallowed just before going to bed, its action would be delayed by sleep. The state (solid or fluid) of the poison would also influence it. The habitual use of narcotics would likewise tend to retard its action. But, even admitting all this, we must still be at a loss to account for many cases of delay in the manifestation of the usual symptoms.

The *external* application of arsenic to abraded surfaces has often been attended with fatal results. Dr. McCready reports a case (Am. Jour. Med. Sci., July, 1851) where a woman applied a mixture of arsenious acid and gin to the head of a child affected with porrigo favosa: it caused death in thirty-six hours. Numerous cases are recorded, both in Europe and in America, of the fatal effects of the application of arsenic to open cancers, by the *cancer-curers.* In some of these cases the poison has been detected after death in the different organs of the body, which had received it by the process of absorption.

Arsenic has also proved fatal when applied to the mucous membrane of the rectum and vagina. In a case reported by Dr. Mangor, a man poisoned three wives in succession, by introducing arsenic into the vagina (Wormley). In at least two of these instances the poison produced its usual symptoms, and death in twenty-four hours. In all the cases above alluded to, of death from the external application of arsenic, the usual characteristic symptoms were noticed, such as the burning and constriction of the throat, the thirst, the vomiting and purging, the great depression, and the various nervous disturbances already mentioned.

Within a few years a number of cases of chronic poisoning have been traced to persons' occupying rooms hung with paper stained with arsenical pigment (Scheele's green): the dust from the paper, becoming detached, and being inhaled into the lungs, occasioned the results mentioned.

Fatal dose.—According to Prof. Lachèse, of Angers, *one to two grains* of arsenious acid may prove fatal to an adult. Dr. Taylor gives a case where two grains of the poison, in the form of Fowler's solution, taken in divided doses during a period of five days, destroyed the life of a woman. The same writer cites another case, reported by Dr. Letheby, in which two and a half grains killed a robust, healthy girl of nineteen years, in thirty-six hours. Much smaller quantities have given rise to alarming symptoms. On the other hand, recovery has taken place from doses of half an ounce, one ounce, and even two ounces of the poison in substance. Dr. A. Stillé (Therapeutics, vol. ii. p. 707) gives the case of a woman who swallowed about a dessertspoonful of the poison immediately after a hearty meal; and although there was no vomiting, and no remedies were administered for an hour and a half, yet within five days complete recovery ensued.

The following remarkable case is reported by Dr. W. C.

Jackson (Am. Jour. Med. Sci., July, 1858). A man aged twenty-eight years took, on an empty stomach, not less than *two ounces* of arsenious acid. Nearly two hours after, there was a slight vomiting, with some traces of the arsenic; but the greater part of the poison was retained in the body for six hours. Great irritability of the stomach then ensued, with a burning sensation in this organ, and in the throat. This condition continued about six hours, after which the patient rapidly recovered.

Fatal period.—The great majority of deaths occur within twenty-four hours. According to Prof. Guy (Forensic Med., p. 449), the average duration of fatal cases is twenty hours. More than half of these terminate within six hours; two-thirds within eight hours; and more than three-fourths within twelve hours. The *shortest* period, with one exception, within which it has proved fatal, is *two* hours, of which three or four instances are on record. Another case, related by Sir R. Christison (On Poisons, p. 240), proved fatal in three hours. Dr. Taylor (Med. Jurisp., 1873, p. 256) relates the most rapidly fatal case yet recorded,—of a youth aged seventeen years, who died from the effects of a large dose of arsenic accidentally swallowed: the symptoms were of a tetanic character, and death took place in *twenty minutes* after swallowing the poison. On the other hand, death may be protracted for months, and even years, the patient experiencing great distress during the whole interval. Belloc reports the case of a woman who died after *two years'* suffering from the effects of arsenic applied externally for the cure of itch. (Cours de Méd. Lég., p. 121.)

Arsenic is not a *cumulative* poison: it is temporarily deposited in the organs—liver, spleen, kidneys, heart, etc.,—after absorption, but it is rapidly eliminated by the urine, and also by the bile; and if the person survives for a certain length of time, the whole of it may be removed from the body, so that none shall be discovered, after death, by chemical analysis.

The exact period required for the elimination of arsenic from the human body has not been definitively settled; although authorities generally agree that fifteen or sixteen days may be regarded as the limit. Orfila held this view. This is on the assumption that there is no suppression of urine, or of the other secretions. The case of Dr. Alexander (Med. Times and Gaz., April 18, 1857) is of great interest in this connection. This gentleman accidentally swallowed a large dose of arsenious acid; and, after suffering from the usual symptoms, he died on the *sixteenth day.* The chemical analysis detected no trace of the poison in any organ of the body, although it was abundantly contained in the food swallowed by the deceased. In this case the arsenic had been completely eliminated in sixteen days. Dr. Maclagan (Ed. Month. Jour., vol. xiv., 1852, p. 131) reports a case in which a woman swallowed half a dessertspoonful of arsenic, but recovered after being under treatment for twenty-five days. The urine was constantly examined, and continued to yield evidence of the presence of the poison up to the twenty-first day.

The rapidity with which arsenic is absorbed and deposited in the tissues is very great. Dr. Taylor (On Poisons, p. 46) found it in the liver in four hours after it had been swallowed. Dr. Geoghegan's observations lead to the conclusion that the liver acquires its maximum of saturation in about *fifteen hours.*

This question of the period of elimination of absorbed arsenic is one of considerable importance in a medico-legal view. For example, if a person who had been taking arsenic in small doses, medicinally, for a length of time, should suddenly die of gastro-enteritis, under suspicious circumstances, the death might be ascribed to poison; and the suspicion would appear to be confirmed by the detection of arsenic in the tissues and organs of the body. Now, if it could be conclusively shown that the deceased had been using arsenic medicinally up to three weeks, or even one month, before death, we are of the opinion that the mere detection of the *absorbed* arsenic, especially if it were absent from the stomach and bowels, would not justify the conclusion that the death had been occasioned by this poison. It is known positively that other metallic poisons, such as antimony, copper, silver, and lead, do remain in the organs many months after they have ceased to be taken; and analogy would lead us to believe that, in some instances at least, the period of the detention of arsenic might be protracted. The case of *Lacoste,* which occurred in France, in 1844, is one in point. The deceased had, for some time previous to his death, been in the habit of using arsenic as a remedy for some cutaneous disease. He died under somewhat suspicious circumstances; and on examination of the body, nine months afterwards, arsenic was found deposited in the soft organs. It was proved that the deceased had taken none of the medicine for a period of fifteen days prior to his death, and the prosecution contended that the arsenic found in the tissues could not be ascribed to this medicine, but must have been some portion of the poison subsequently administered, and therefore that the prisoner was guilty of the crime. No arsenic was found in the *free* state in the bowels, and the stomach does not appear to have been examined! Under these circumstances, the prisoner was very properly acquitted. The small quantity of the poison found in the organs was considered quite consistent with the theory of medicinal administration (*vide* p. 28).

Arsenic is known to be deposited in the bones, but not in the hair; neither in the feathers of birds (Taylor).

Post-mortem appearances.—These are to be especially noticed as they are presented in the stomach and bowels. They are generally well marked in proportion to the size of the dose, and the length of time elapsing after taking the poison. Arsenic seems to have a specific effect on the stomach, no matter how it may have entered the system—whether swallowed, or injected into the rectum or the vagina, or inhaled into the lungs, or introduced subcutaneously. Accordingly, in the generality of cases we find the mucous membrane of the stomach exhibiting evidences of active inflammation. Sometimes the whole surface is of a deep crimson color; at other times it is of a deep brownish red; again, it is interspersed with dark patches or lines of effused blood, which have been mistaken for gangrene. Occasionally it is much thickened and corrugated. When the arsenic has been taken in substance, it is common to find several patches, varying in size from one inch to three inches, consisting of a tough white or yellowish paste of arsenious acid mixed with lymph and mucus firmly adherent to the inflamed membrane, and forming so many centres of intense inflammation. White spots of arsenious acid are often found between the rugæ; and sometimes yellow points of the sulphide, in cases where the examination has been made a long time after death, and where decomposition has advanced. Tardieu very properly cautions us not to mistake, for these, certain little white and yellow specks, frequently found on the inner walls of the stomach, and consisting merely of albumen and fatty matters.

Ulceration of the stomach is of rare occurrence; but it has been found as early as ten hours after the poison had been taken. We have seen a case, where the examination was made about four months after death, in which there was an ulcer about a quarter of an inch in diameter near the greater extremity: it was surrounded by a deep zone of dark, effused blood, and it had penetrated down to the peritoneal coat of the organ. Perforation of the stomach is still more uncommon; although a few instances are reported.

The inflammation usually extends down the alimentary canal, involving the duodenum and portions of the ileum. It is apt to be more decided about the cæcum and rectum. The œsophagus also is occasionally the seat of inflammation, and more rarely the mucous lining of the mouth, fauces, and tongue. The lungs and brain have occasionally been found congested, and in some cases the bladder has exhibited signs of inflammation; but none of these are sufficiently characteristic to be of any medico-legal importance. The blood is usually liquid, and of a dark color. The most remarkable fact connected with the post-mortem signs, is the occasional absence of all traces of inflammation, and of every other characteristic change; and this, too, where there had been well-marked and violent inflammatory symptoms before death.

Another point worthy of observation is the *antiseptic* power of arsenic over the body. As a rule, the body will be found in a remarkable state of preservation, even many months after death from arsenical poison. This preservative influence is more particularly noticed in the internal parts, where the poison has come into direct contact with the organs. In such cases, a most noticeable fact, in making the autopsy, is the absence of all true cadaveric odor: in its stead a peculiar smell is perceived, which is described as resembling that of old cheese. Very generally also, in such cases, on opening the abdominal cavity, the contents will present a decidedly yellowish appearance,—the color being brighter in spots scattered along the stomach and intestines. This yellow color is due to the yellow sulphide (orpiment), which has been produced, in the process of putrefaction, by the action of sulphuretted hydrogen on the arsenious acid. Christison alludes to this as "the effect of a chemical test applied to the poison by nature." The time at which this change takes place varies: Dr. Taylor has found it as early as twenty-eight days after death, though, of course, the greater the length of time, the more complete would we naturally expect the conversion to be.

The length of time that the preservative effects of arsenic will last upon a human body seems to vary. It is by no means unusual to find the body in a good state of preservation many months, or even years, after burial. Of course, in the latter case, many portions might be more or less decomposed; but the point of medico-legal interest is, that the poison can thus be discovered years after its administration. Dr. Webster found it in a body *fourteen* years after death (Boston Med. and Surg. Jour., vol. xxxix. p. 489).

It is fortunate for the ends of justice that arsenic not only preserves the stomach where surrounding parts are in a state of decay, but that even the characteristic marks of inflammation may be present after several months of interment.

It is proper to state that the antiseptic power of arsenic is not always witnessed in the dead body. In fact, in some cases the process of putrefaction seemed to advance with increased rapidity. Besides, it must be remembered that the body is sometimes unusually preserved in ordinary cases of death. Nevertheless, the general fact of the antiseptic power of arsenic cannot be disputed. It is, therefore, evident that the expert witness would not always be justified in asserting that, because the body had resisted putrefaction for an unusual length of time, this preservation was due to the presence of arsenic, since it may really be attributable to other causes (*vide ante*, p. 41). This question may assume very considerable importance in a trial, where the defense takes the ground that any arsenic found in the body a long time after burial, has resulted from post-mortem imbibition of the poison, *which had been secretly introduced into the stomach after death*, and not from ante-mortem poisoning. If, in such a case, it should happen that the body had been better preserved than usual, the mere *fact* of such preservation could not, as we have seen, be *positively* ascribed to the presence of arsenic (*ibid.*).

Treatment.—If the patient is not already vomiting freely, a prompt emetic should be administered, such as a combination of sulphate of zinc and ipecacuanha; or, in the absence of this, some warm mustard and water. The vomiting should be assisted by the free use of warm demulcent drinks. After this, the *hydrated sesquioxide of iron*, in the moist state, should be freely exhibited. This substance may be regarded as the best-known antidote for arsenic. It most probably acts by combining with it, so as to form the insoluble arsenate of iron. This antidote should be administered in large excess, and, as already stated, in *the moist state*. It may be extemporaneously prepared by diluting the *tinct. ferri chloridi* of the shops with water, and adding *aqua ammoniæ* to precipitate the sesquioxide. The precipitate should be thoroughly washed, and given in tablespoonful doses. After the employment of the antidote, a dose of castor oil may be administered, to carry the resulting compound through the bowels.

In relation to the efficacy of this antidote, we believe there can be no doubt. Numerous cases are reported attesting its value in poisoning from arsenic. Prof. Wormley (Micro-Chemistry of Poisons, p. 247) gives the results of twelve experiments made upon dogs; these had previously, or simultaneously, taken a dose of arsenious acid which had by a prior experiment been proved to be a fatal dose: in every instance the animal entirely recovered.

The freshly precipitated *hydrate of magnesia* has also been recommended as an antidote. The treatment of the resulting inflammation and of the other symptoms should be conducted on general principles.

Chemical analysis.—I. *In the solid state.*—(1) A small quantity of the white powder placed on platinum-foil and heated in a spirit-lamp is entirely volatilized, giving off a white, inodorous vapor. (2) Slowly heated in a narrow reduction-tube, it sublimes without melting, and is deposited in the form of a white ring of brilliant octahedral crystals, visible by a good magnifier, or a low power of the microscope. (3) Moistened by liquor potassæ, it undergoes no change of color,—which distinguishes it from either calomel or corrosive sublimate. (4) Moistened by sulphide of ammonium, and allowing the excess of ammonia to pass off, a yellow sulphide of arsenic remains. (5) Mixed with some reducing agent, such as charcoal, black flux, cyanide of potassium, or ferrocyanide of potassium, and heated in a reduction-tube, the metal is reduced and volatilized, condensing in the form of a metallic ring or mirror, of great brilliancy, and of a steel-gray color. (6) The sublimed metal in the state of vapor has the odor of garlic.

The two processes of *sublimation* and *reduction* require a little fuller description. In order properly to develop the characteristic crystalline appearance of the sublimate of arsenious acid, experience has shown that the white vapor must be received on a warm surface. If the surface is perfectly cold, the deposit is apt to be amorphous and imperfect. The experiment may be performed either with the reduction-tube, or with the glass disk, as recommended by Prof. Guy. The reduction-tube should be composed of thin, hard German glass, about three inches in length, and one-sixteenth of an inch in diameter. The inside of the tube must be perfectly clean, and free from moisture. The powder being introduced, and the tube wiped out, if necessary, it is first to be gently heated in the flame of the spirit-lamp a little distance above the contents, after which, the flame is to be directly applied to the bottom of the tube, until the sublimation is accomplished. Dr. Guy's method (Foren. Med., p. 383) consists in placing the powder, perfectly dried, in a narrow glass tube about three-quarters of an inch in length, and having its mouth covered over with a warm glass slide or disk. On applying heat to the bottom of the tube, a beautiful deposit of crystals takes place on the glass cover. The flat surface of the latter renders it peculiarly favorable for microscopic examination.

It should be remembered that other substances besides arsenious acid will undergo sublimation by heat and will be deposited in white rings, such as calomel, corrosive sublimate, oxalic acid, and salts of ammonia. But most of these substances melt before subliming, and *none of them condenses in the form of octahedral crystals.* It will be noticed, then, that it is the sublimate composed of octahedral crystals that is the unequivocal proof of the presence of arsenious acid; and the student will do well to familiarize himself with the appearance of arsenical sublimates, obtained after the methods just described.

The crystals thus obtained, especially if the original quantity is very minute, are exceedingly small, but still perfectly characteristic. With a power of one hundred diameters, we may determine the angles of a crystal that does not exceed one eight-thousandth part of an inch in diameter; and with a power of two hundred and fifty, crystals measuring only one fifteen-thousandth of an inch may be satisfactorily determined (Wormley).

The reduction process consists in heating the arsenious acid, along with some reducing agent, in a reduction-tube: the reduced metal rises in vapor, and is deposited, in the form of a bright, steel-gray-colored ring, on the cool part of the tube. In this experiment, there are frequently two rings in the tube: the upper and longer ring has a brown color, and appears to be a mixture of finely-divided metallic arsenic and arsenious acid; the lower ring is small, and has a bright mirror-appearance, like polished steel; it consists of the pure metal. The inner surface of the latter sublimate presents a bright crystalline appearance.

Practically, one of the best methods for effecting the reduction of arsenious acid, and one which is equally applicable for the sulphides and the arsenites, is by means of the perfectly dry ferrocyanide of potassium,—as proposed by Dr. E. Davy, of Dublin (Chem. News, vol. iii. p. 288). This salt is preferable to the cyanide, in that it is not deliquescent. After being thoroughly dried at a temperature of 212°, it should be mixed with the arsenious acid, in the proportion of six or eight parts of the former to one of the latter, and introduced into the reduction-tube. The mixture blackens before fusing.

When the quantity of the arsenious acid is extremely minute, the tube should be proportionately small. The best mode of procedure, under such circumstances, is to use a thin tube not over three inches long and about one-sixteenth of an inch in diameter. After introducing the mixture into the bottom of the tube, the latter should be thoroughly wiped out with a piece of filtering-paper properly rolled up: the tube should then be heated at a little distance above the mixture by a blowpipe flame, and drawn out into a contracted neck. After this has sufficiently cooled, and before it is *entirely* cold, the heat should be applied to the bottom of the tube, when the metallic sublimate will become very apparent in the contracted neck. According to Prof. Wormley, the one-thousandth of a grain of arsenious acid, when treated in this manner, will give a very satisfactory metallic sublimate, which, upon resublimation, farther up the neck of the tube, will furnish several hundred octahedral crystals of arsenious acid, many of them measuring one-thousandth of an inch in diameter.

Although the metallic crust, obtained in the manner just described, is positive proof of the presence of arsenic in the original mixture, still, a witness, on a trial for poisoning, would not be justified in so affirming, without proceeding a step further. Compounds of mercury, cadmium, selenium, and tellurium may, under similar circumstances, yield sublimates. These, however, may easily be distinguished from the arsenical sublimate, even by the naked eye, though still better by the microscope, which exhibits them in the form of globules or drops. But there are other still more confirmatory tests: (1) the arsenic mirror is wholly soluble in a solution of hypochlorite of soda; (2) it is also soluble in warm nitric acid; and when this solution is evaporated to dryness by heat, and the residue is touched by a drop of a strong solution of nitrate of silver, a brick-red color is produced, due to the formation of *arsenate* of silver. If the closed end of the tube be broken off, and heat be applied to the sublimate, it will readily volatilize, and the vapor combining with the oxygen of the air will condense in the octahedral crystals of arsenious acid. Or, the portion of the tube containing the crust, having been separated by the file, may be broken up and introduced into another reduction-tube: when this is properly heated, the characteristic crystalline deposit takes place.

The conversion of the metallic crust into the octahedral crystals of arsenious acid by resublimation is a highly satisfactory proof of the presence of this poison; but we may proceed a step further, and dissolve these crystals in a few drops of water, and subject the solution to the liquid tests (see *post*).

It has been objected that a crust of charcoal, or the employment of a reduction-tube containing lead, might occasion an error: it is difficult to understand how a practiced eye could possibly mistake them; but all room for doubt will certainly be removed by resubliming the suspected crust: the crystalline deposit can occur *only* if this crust is arsenical.

II. *The liquid tests.*—These are two in number, *viz.*, the *ammonio-sulphate of copper*, and the *ammonio-nitrate of silver.* These tests should both be prepared at the time they are required for use. The former is made by cautiously adding solution of ammonia to a somewhat dilute solution of sulphate of copper, until the precipitated oxide is *almost* redissolved. When this reagent is added to a solution of arsenious acid, it throws down a light-green, amorphous precipitate of arsenite of copper, known as *Scheele's green.* This precipitate is very soluble in ammonia and in free acids. From very dilute solutions of arsenious acid, the precipitate does not assume its characteristic color until it has stood for some time.

The *ammonio-nitrate of silver* is prepared by cautiously adding a weak solution of ammonia to a solution of nitrate of silver, until the precipitated oxide of silver is *nearly* redissolved. This reagent throws down from a solution of arsenious acid a canary-yellow precipitate—*arsenite of silver*

—which is freely soluble in ammonia, and in nitric and acetic acids.

The above liquid tests are conclusive only when applied to a solution of *pure* arsenious acid: in organic solutions they are valueless as proof of the presence of arsenic, since certain organic substances yield with each of the liquid tests, colors similar to those caused by arsenic. Phosphoric acid and the phosphates also give to a solution of nitrate of silver a delicate yellow color. Hence the mere production of these colors is not of itself proof of the presence of arsenic. The danger of placing an undue reliance on these color tests is shown in the celebrated *Donnall case*, which was tried in England in 1817. Here, the charge of poisoning by arsenic was sustained chiefly by the results afforded by the two liquid tests upon the boiled contents of the stomach: the yellow and green precipitates were thrown down; but it was subsequently shown that a decoction of onions would produce a precisely similar green color with the copper test, as the one actually obtained by the analyst. (See Beck's Med. Jurisp., vol. ii. p. 580.)

The liquid tests can, however, be easily confirmed, as follows: (1) by heating either of the dried precipitates (arsenite of silver and arsenite of copper) in a reduction-tube, either alone, or with a reducing agent; a sublimate of octahedral crystals of arsenious acid will be yielded in the first case, and a mirror of metallic arsenic in the second. (2) If a small quantity of the blue ammoniacal solution of the arsenic is poured over a crystal of nitrate of silver, a film of yellow arsenite of silver will appear around the crystal. The silver and copper tests may thus be used to confirm each other.

III. *The sulphuretted hydrogen test.*—Sulphuretted hydrogen gas, and its solution in water, both give with solutions of arsenious acid slightly acidulated, a bright yellow amorphous precipitate of tersulphide of arsenic, or *orpiment*, which is soluble in the alkalies, but insoluble in cold hydrochloric acid, and only partially soluble in the boiling strong acid; hot nitric acid dissolves and decomposes it to arsenious acid. From very dilute solutions of the poison, the precipitate does not separate until the excess of the sulphuretted hydrogen is expelled by heat, or on exposure to the air. In all cases, a gentle heat favors the complete separation of the precipitate.

There are other substances besides arsenic which give yellowish precipitates with sulphuretted hydrogen, such as *cadmium*, *selenium*, and *tin:* the color of the *antimonial* precipitate is orange-red. The sulphide of cadmium is in appearance very similar to the sulphide of arsenic; but they differ entirely in that the cadmium-sulphide is soluble in hydrochloric acid and insoluble in ammonia, while it is just the reverse with the arsenic-sulphide. The bisulphide of tin, when dried, has a dull yellow color; it is partially soluble in cold hydrochloric acid, but sparingly soluble in ammonia. The sulphide of selenium, besides being of very rare occurrence, soon changes to an orange-red color, and is insoluble in ammonia.

The confirmatory proofs of the sulphide of arsenic are: (1) when thoroughly dried, and mixed with dry ferrocyanide of potassium, black flux, or cyanide of potassium and carbonate of potash, and heated in a reduction-tube, it yields the characteristic sublimate of metallic arsenic. In a small reduction-tube properly drawn out, satisfactory results may be obtained with a precipitate from the one-thousandth of a grain of arsenious acid. (2) Boil the hydrochloric acid solution of the yellow precipitate with a strip of bright copper-foil, and proceed as directed for *Reinsche's test* (see *post*): no other substance, thus treated, will yield the characteristic octahedral crystals. (3) Dissolve the precipitate in a little hot nitric acid, evaporate to dryness, and add a drop or two of solution of nitrate of silver: it will yield the brick-red colored arsenate of silver.

IV. *Marsh's test.*—This valuable test was first proposed by Mr. Marsh, of Woolwich, about the year 1835. The principle involved in it is, that when an arsenical compound comes in contact with *nascent* hydrogen, the arsenic combines with the latter, to form *arsenetted* or *arsenuretted hydrogen*—a gas possessing peculiar properties, by which the presence of the poison may with certainty be recognized. The original *Marsh's apparatus* consists of a U-shaped glass tube, one leg of which is longer than the other. The longer one is open;

the shorter one is closed by a stop-cock furnished with a nozzle, terminating in a minute bore. Hydrogen gas is generated by pouring dilute sulphuric acid through the open end of the tube, upon a fragment of pure zinc; the gas evolved gradually accumulates in the short leg of the tube, and escapes on opening the stop-cock. If the arsenious acid be now introduced along with the contents of the tube, arsenuretted hydrogen will be generated, and will escape in a small jet on opening the stop-cock. On ignition, it will yield the characteristic results. A cheaper and more simple form of apparatus may be used, and one which is preferable in toxicological investigations, where *new* apparatus should be employed in every new analysis. It consists simply of the ordinary hydrogen-bottle, fitted with two glass tubes, one a straight funnel-tube, for introducing the materials, the other one bent at right angles, for the delivery of the gas. To the latter may be attached a drying-tube, with the proper arrangement for burning the gas, as it escapes.

In performing this experiment, certain precautions are necessary. In the first place, the zinc and sulphuric acid must be proved to be free from contamination with arsenic. It has already been stated that both of these substances, as found in commerce, are very apt to contain arsenic as an impurity. Secondly, care should be exercised that all the atmospheric air has time to escape from the gas-bottle before lighting the jet, otherwise the mixture of the air and hydrogen would occasion an explosion, which might be attended with unpleasant consequences. With these precautions, the experiment is performed as follows: fragments of pure zinc are put into the bottle, and a mixture of pure sulphuric acid and water, in the proportion of one measure of the former to four of water, is poured through the funnel-shaped tube; effervescence immediately takes place from the escape of hydrogen gas, which passes off through the other bent tube; this latter is connected, by means of a cork, with the drying-tube, which is filled with fused chloride of calcium, or with cotton moistened with strong sulphuric acid: the drying-tube is connected by a piece of india-rubber tubing with the reduction-tube; this should be of hard glass, without lead, about an eighth of an inch in diameter, several inches long, contracted in two or three places, and terminated in a turned-up, drawn-out point, for ignition of the gas. Several of these tubes should be at hand.

When sufficient time has been allowed for the escape of all the atmospheric air, the jet may be lighted: it will burn with a scarcely-perceptible flame, if it be pure hydrogen gas. The purity of the materials is next to be tested by applying the flame of a large spirit-lamp, or a Bunsen-jet, under the horizontal delivery-tube, until it is heated to redness, just behind one of the contracted portions: if no metallic deposit or stain occurs in the contracted part of the tube, the materials may be considered free from arsenic. Or, if on holding a clean, dry, white porcelain lid over the ignited jet, so as to depress the flame, no metallic deposit or spot is formed, the same conclusion may be held.

A small quantity of the arsenical solution is next introduced through the funnel-tube: its decomposition immediately commences, resulting in the formation of *arsenetted hydrogen;* this is recognized by the following characteristics:

1. *The ignited jet.*—The moment the arsenic combines with the hydrogen, an obvious change is perceived in the flame, which increases in size, and acquires a sickly bluish tint; and, unless the arsenic is in very minute quantity, it evolves the white fumes of arsenious acid, and emits a peculiar alliaceous odor. If these fumes be received into a short, wide test-tube, or on an inverted watch-glass, they will condense into a white powder, which is sometimes crystalline (octahedra), and which can be proved to be arsenious acid by any of the tests already described.

If the flame be made to strike against a piece of cold glass, or white porcelain, held horizontally, it yields a deposit of pure metallic arsenic, in the form of a brilliant steel-gray or brownish spot. By changing the position of the cold surface, numerous deposits of this nature may be obtained. The exact appearance of these spots depends very much upon the quantity of arsenic present, and on the character of the flame. The jet should not be too large, and should burn with a clear, steady flame: to this end, the evolution of gas should be moderately slow. When the flame is very small and round, the surface should be held very near its base; but when the flame is pointed, or conical, the porcelain should be held at about its upper third. The color of the spots varies: sometimes they present a very brilliant steel-gray appearance; again, they may be of a dark, brownish-black color; and sometimes they may even exhibit a copper hue; but never a black, *sooty* appearance.

These spots may be identified as arsenical by the following tests: (1) they are immediately soluble in a solution of hypochlorite of soda or lime; deposits of antimony (which most resemble them) are not thus soluble. (2) When touched with a drop of sulphide of ammonium (which instantly dissolves antimony), they do not immediately disappear, but require some time for solution. When the ammoniacal solution is evaporated to dryness, a *bright yellow* spot is produced (sulphide), which is readily soluble in ammonia, and insoluble in cold hydrochloric acid; under the same conditions, antimony yields an orange-red residue, which is insoluble in ammonia, but soluble in strong hydrochloric acid. (3) The deposits from both metals are dissolved in a drop of warm nitric acid, and yield on evaporation white residues. When, however, these are touched with a drop of strong solution of nitrate of silver, the arsenical spot assumes a brick-red color, while the antimonial spot remains unchanged. (4) The antimonial spot is generally darker and less lustrous than the arsenical: if, however, it is very thin, it may present an appearance precisely similar to that of the arsenic spot.

There are some other fallacies connected with this test, which deserve a passing notice. Organic matter under certain circumstances, certain combinations of iron and zinc, phosphorus and sulphur, may, under the above conditions, yield stains somewhat resembling those of arsenic. But none of these substances will yield a succession of deposits; and none will yield a single spot possessing the characters above enumerated as belonging to the arsenical deposit. It is to be remembered that the evidence of the presence of arsenic is not based alone upon the obtaining of a spot, or spots, by Marsh's test, *but upon the identification of these spots*, in the manner above described.

The delicacy of this process is such, that one five-thousandth of a grain of arsenious acid may by this means be detected.

2. *Decomposition of the gas by heat.*—Berzelius modified the original experiment of Marsh by decomposing the arsenetted hydrogen gas by means of heat, as it passes along the horizontal tube. For this purpose, the flame of a large spirit-lamp is placed under the tube, about three-quarters of an inch on the inside of one of the contractions. When the tube becomes red-hot, a small quantity of the arsenic solution is poured into the flask; as the resulting arsenetted hydrogen passes through the red-hot tube, it is decomposed, and a bright mirror of metallic arsenic is deposited in the contracted part of the tube. A series of such mirrors may be obtained by applying the heat to successive portions of the tube. These deposits are highly characteristic, though they may vary somewhat in color from a steel-gray to almost a copper hue: they serve as admirable illustrations of the *presence of the metal*, which can be exhibited in court, as positive proof of the detection of the poison. Moreover, they may be resublimed into arsenious acid, and this again can be subjected to the liquid tests.

The delicacy of this modification is even greater than that by which the gas is burned in a jet for the production of the spots. Unless the quantity of the poison be exceedingly small, it is always possible to obtain *both* results by Marsh's process.

The only fallacy to which this experiment is liable is the presence of antimony. *Antimonetted hydrogen*, treated as above, will give a deposit of metallic antimony, which might possibly be mistaken for one of arsenic. They may, however, be distinguished as follows: since the antimonetted hydrogen is decomposed at a lower temperature than the arsenetted, the deposition of metallic antimony takes place just over the heated spot, or rather on both sides of it, whilst the arsenic is always deposited half an inch or three-quarters in advance of the flame. Again, the antimonial mirror has usually a duller black appearance than the arsenical, although sometimes this distinction cannot be perceived. Again, there is a marked difference in the volatility of the two deposits: if the flame be applied to the arsenic mirror, it very soon disappears, and condenses farther on in the tube in the characteristic octahedral crystals; the antimony mirror is more slowly affected, requiring a higher temperature, and yields a white, amorphous deposit, quite near the point to which the heat was applied. The deposits of the two metals may be further distinguished, as before stated, by the action of sulphide of ammonium, hypochlorite of soda, and nitric acid (*ante*, p. 235).

The action of *dry sulphuretted hydrogen gas* upon the deposits of these two metals affords a further ground of distinction. If a stream of this gas be passed through a tube containing the arsenic crust, and the crust be chased in the opposite direction to the stream of gas by the flame of a spirit-lamp, it is rapidly vaporized and converted into the yellow tersulphide, which is deposited a little in advance of the flame. The antimonial crust, treated in a similar manner, deposits a reddish-brown, nearly black, tersulphide of antimony, but much nearer the flame, and requiring also a higher heat. If the tersulphide of antimony be exposed to a slow current of dry hydrochloric acid gas, it rapidly disappears, whilst the tersulphide of arsenic, similarly treated, is unaffected. (Pettenkofer and Fresenius.)

3. *Decomposition by nitrate of silver.*—The nature of the arsenetted hydrogen may be further proved by the action of nitrate of silver. If the gas be conducted into a solution of this salt, it blackens immediately; double decomposition takes place, resulting in the production of arsenious acid, which remains in solution, and metallic silver, which falls as a black precipitate. The filtered solution will contain, besides arsenious acid, free nitric acid, and any excess of nitrate of silver. On neutralizing with ammonia, this solution will yield a yellow precipitate of arsenite of silver (see p. 230). Should no nitrate of silver happen to be in the solution, a little of that salt must be added, after the ammonia. Again, after removing any excess of nitrate of silver from the solution

by hydrochloric acid, and filtering, if the filtrate be treated by sulphuretted hydrogen, it will yield the characteristic yellow precipitate of the tersulphide of arsenic. Or, instead of using sulphuretted hydrogen, the filtrate may be examined by Reinsch's test (see *post*). Or, finally, if the filtrate be evaporated to dryness, the arsenic will remain as white *arsenic acid*, which, when moistened with a strong solution of nitrate of silver, assumes a brick-red color.

This reaction with nitrate of silver is extremely delicate: according to Wormley (Micro-Chem. of Poisons, p. 291), the one-hundred-thousandth of a grain of arsenious acid may be thus detected.

It must, however, be remembered that it is not the mere production of a *black color* with nitrate of silver that furnishes the proof required, since several agents will cause this,—for example, antimonetted hydrogen, sulphuretted hydrogen, and phosphuretted hydrogen. As regards the antimonetted hydrogen, the resulting black precipitate contains the whole of the antimony in combination with the silver, as an antimonide of silver: consequently, the filtered solution should give none of the reactions of arsenic.

V. *Reinsch's test.*—In this process, the liquid containing the arsenic, or the solid dissolved in distilled water, is boiled with one-sixth to one-eighth of pure hydrochloric acid, and a small strip of bright copper-foil is then introduced. The presence of a minute quantity of arsenic is indicated by the copper immediately becoming tarnished from the deposition of metallic arsenic upon its surface. The *color* of the deposit depends upon the amount of arsenic present: if the quantity be large, the film upon the copper has a dark iron-gray tint, sometimes almost black; this is apt to scale off, especially if the liquid be long boiled. If the arsenic be in very small quantity, the polished copper will merely acquire a faint violet or bluish tint. The deposit upon the copper is always affected by the degree of dilution, and where the quantity of water present is large, it may require boiling for half an hour before the deposition upon the copper becomes visible.

This reaction is extremely delicate, and the results are very satisfactory. Certain precautions must be observed to secure success: first, the purity of the hydrochloric acid must be certain; this can, happily, be demonstrated by the test itself. It is only necessary to boil some of the acid, diluted with six or eight parts of pure water, upon a slip of copper-foil: if the latter is not tarnished after the lapse of fifteen or twenty minutes, we may be certain of the absence of arsenic. It is also important that the copper should have a bright surface: this is effected by means of emory-paper. The purity of the copper-foil should likewise be secured. If the copper, when boiled with the acid liquor containing the arsenic, is not dissolved, and does not impart a green color to the liquid, it may be considered pure. It is only when the copper is dissolved in the liquid which is being tested, that the impurity of the metal can affect the result.

According to Dr. Taylor (Med. Jurisp., 1873, p. 261), copper may be best tested for arsenic by the simple method of Mr. Abel: Add to pure hydrochloric acid, diluted with six parts of water, one or two drops of a weak solution of perchloride or persulphate of iron: boil the acid liquid, and introduce into it the copper well polished: if it contains arsenic, it soon becomes tarnished; if pure, it retains its brightness.

It is best not to employ too large a surface of copper in the first instance, but successive strips should be introduced as each one becomes completely coated. By this means the whole of the arsenic may be withdrawn from a solution. Another point to be noticed is, not to remove the copper too soon from the boiling liquid; for if the quantity of arsenic present be very small, it may require a considerable time before the deposit takes place. In doubtful cases the boiling should continue for half an hour, before deciding positively as to the absence of arsenic. But, on the other hand, if the copper be kept in for an hour or longer, it may acquire a dingy appearance, from the action of the acid and air exclusively.

Fallacies.—Various other metals besides arsenic are deposited upon copper under the same conditions, viz., antimony, mercury, silver, tin, bismuth, gold, platinum, and palladium; so also organic matter, *especially if it contain sulphur.*

The antimonial coating has usually a well-marked violet hue, while the deposits of mercury, silver, and bismuth have generally a bright silver appearance, and that of gold a yellow color. Under certain circumstances, however, they may all resemble the deposit caused by arsenic. Hence it would be very unsafe to rely solely upon the *color* of the film, in Reinsch's test. The corroborative proof consists in subjecting the coated copper-foil to heat, in a reduction-tube. The arsenic will volatilize, and, meeting with oxygen, will condense higher up in the tube as arsenious acid, recognizable by the octahedral crystals. The only other metals which could volatilize under such circumstances are antimony and mercury; but the sublimate from mercury consists of spherical metallic globules, easily distinguished by the microscope; and that from antimony, although white, is either amorphous or granular, or else in fine acicular crystals; the deposit, moreover, occurs nearer to the copper, and requires a higher heat for its production, than in the case of arsenic.

Dr. Wormley (Micro-Chem. of Poisons, p. 274) alludes to the fact that when complex organic mixtures acidified by hydrochloric acid are boiled for some time in contact with metallic copper, the metal will be coated with a very perceptible stain, and yield by heat an amorphous sublimate, which sometimes contains acicular crystals, consisting apparently of a compound of copper. We have frequently verified the above observation in relation to the action of organic matter. We have experimented upon a mixture of tincture of gelsemium, chloral, and milk, with hydrochloric acid, and obtained a decided stain upon copper-foil, which might easily be mistaken for an imperfect stain of either arsenic or antimony; moreover, on subjecting the copper to sublimation, a white, amorphous deposit took place in the tube. From what has just been stated, it follows, that for the complete verification of Reinsch's test, in the case of arsenic, *nothing less than the production of the octahedral crystals, and their subsequent identification*, should be admitted in any medico-legal investigation.

In all cases, the size of the reduction-tube should be proportioned to the amount of arsenic examined by the above test. If the coating upon the copper is very decided, a single slip half an inch square will suffice to give the characteristic crystals by sublimation; if the deposit is very slight, several such pieces of copper should be used at once. Some caution is to be observed in preparing the copper for sublimation. After the deposition of the arsenic, the slips should be withdrawn from the liquid and washed thoroughly in distilled water, and then completely dried by bibulous paper. Moisture should be carefully avoided.

Interferences.—Certain substances, if present in the arsenical solution, may prevent the deposition of arsenic on copper, as *a chlorate—e.g.* chlorate of potassa, *binoxide of manganese*, and other substances that decompose hydrochloric acid, giving rise to chlorine. Free nitric acid likewise, when present in notable quantity, or when the solution is much concentrated, will act similarly. In all the above cases, the copper is attacked, and partially dissolved, giving a bluish or greenish-blue color to the solution.

It may be remarked, in conclusion, that Reinsch's method possesses many advantages: the facility with which it may be applied, the fact that it can be used in complex, highly-colored organic mixtures, and the complete separation by it of the poison from such mixtures, entitle it to the highest consideration of the toxicologist.

VI. *Bloxam's method.*—The principle involved here is the same as that in the process of Marsh—the formation of arsenetted hydrogen by the action of nascent hydrogen on arsenious acid. Prof. Bloxam employs electrolysis as the means of decomposing the water and liberating hydrogen; thus dispensing with zinc entirely. The arsenetted hydrogen is made to pass through a horizontal glass tube, in which it is decomposed by heat, as in Marsh's apparatus (see Guy's Forensic Med., p. 441). This method has been found to be very delicate and satisfactory. One precaution is, however, necessary: if the poison exists in the form of *arsenic* acid, its decomposition does not take place; it must first be deoxidized by means of sulphurous acid, or a soluble sulphite.

There are some other reagents, of inferior importance, which may be used to detect arsenious acid: these are *lime-water*, *iodide of potassium*, *bichromate of potassa*, and *sulphate of copper with potassa;* but, as none of these are characteristic, they need not be further noticed.

Separation from organic mixtures.—As arsenious acid is only sparingly soluble in water, and even less so in organic mixtures, it may frequently be discovered in lumps, or particles, by diluting the mixture with distilled water and allowing the solid particles to subside,—using a lens, if necessary. Any white particles should be carefully removed, washed in pure water, and then in ether, to remove any adhering fat, and examined as recommended for solid arsenic (see p. 227). Some of the particles may also be dissolved in water, and subjected to the liquid tests (see p. 230).

Whether the poison is thus discovered or not, the solids and liquids are next to be intimately mixed, together with the addition of hydrochloric acid, and gently boiled for ten or fifteen minutes. When cool, the mixture is to be filtered through muslin, and concentrated by evaporation over a water-bath. A portion may now be tested by Reinsch's process, fresh slips of copper being added, as long as they acquire a tarnish. If the copper does not receive a coating immediately, the boiling should continue nearly to dryness, before concluding that no arsenic is present. In employing this process, it must be remembered that it is the obtaining of the characteristic octahedral crystals on subliming the copper strips, that constitutes the true evidence of the presence of arsenic.

If this method fails to indicate the poison, it may be considered as absent, unless the presence of some interfering substance can be shown. Should it seem desirable, another portion of the filtrate may be tried by Marsh's test; and a third portion by sulphuretted hydrogen: the resulting sulphide, when purified, affords good data for estimating the original quantity of the poison.

Vomited matters.—These are to be carefully collected, and searched for any particles of the poison. The mass is then diluted with distilled water strongly acidulated with hydrochloric acid, and kept boiling for about twenty minutes.

After cooling, it is filtered, the filtrate concentrated, and then examined in the manner described above.

It should be remembered that a failure to detect poison in the matters vomited is not, of itself, conclusive evidence that the poison had not been swallowed, since it might have been absorbed into the system before the vomiting occurred, or even might, if in the solid state, adhere tenaciously to the walls of the stomach, and not be rejected in the act of vomiting.

The stomach and its contents.—Before these are subjected to chemical analysis, they should be carefully examined for the presence of any solid particles of the poison, as already mentioned. The stomach should be placed upon a perfectly clean, large porcelain plate (a new one is preferable), and after being cut open through its smaller curvature, should be completely spread out for minute inspection. This is the proper occasion, also, for noticing the pathological changes of this organ. The contents may now either be examined separately, or, as is more usual, the stomach may be cut up into small pieces (using for this purpose a *new* pair of scissors, or one known to be absolutely clean), and added to the contents. Distilled water should be added, if the mass is too viscid, and mixed with about one-eighth of its volume of pure hydrochloric acid, and heated nearly to 212° F., until the organic matters are completely disintegrated. After cooling, the mixture is to be thrown upon a muslin strainer, and the matters upon the strainer washed several times with pure warm water. The strainer and its contents may be retained for future examination, if necessary. The filtrate should be concentrated by a moderate heat, and, after cooling, be filtered through paper.

Reinsch's test may now be applied to a given portion of the filtrate, as a trial test, care being taken to have the acid liquid boiling, before introducing the copper slips: these should be successively added as long as they receive a deposit. The copper pieces, after being thoroughly washed and dried, are heated in a reduction-tube, and the resulting sublimate examined after the manner already mentioned. Another given portion of the filtrate should be examined by sulphuretted hydrogen. For this purpose, a stream of the gas prepared by the action of pure dilute sulphuric acid on pure sulphide of iron (artificial) should be slowly passed through the liquid slightly warmed, for several hours,—in some instances, for twenty-four hours. It should then be gently warmed, and allowed to stand quietly until the supernatant liquid is perfectly clear. The precipitate thus obtained will have, if arsenic be present, a yellowish color (not the bright yellow of *pure* sulphide), due to the mixture of organic matter and free sulphur, which are always present in greater or less quantity.

It is a point of great importance, in toxicological investigations, to give due attention to the nature of this precipitated sulphide. A careless and hasty conclusion often leads to error in this regard. We have known such precipitates to be mistaken for those of metallic sulphides (especially in the case of arsenic and antimony) when they contained no trace of a metal, but consisted solely of organic matter and free sulphur. In fact, even if arsenic or antimony be present, and the impure sulphide which is thrown down be separated by filtration, and sulphuretted hydrogen gas be again passed through the clear filtrate, a *second* precipitate will be formed, of a dirty yellowish-brown color, composed exclusively of organic matter and free sulphur.

There is no doubt that certain kinds of organic matter will yield, under such conditions, more suspicious-looking precipitates than others; but the fact that *they are thus produced* should be sufficient to make us extremely cautious in our inferences as to the *nature* of such a precipitated sulphide simply from its *color:* a further examination, in all cases, becomes necessary.

The precipitate produced by the sulphuretted hydrogen is collected upon a small filter, washed while still moist, and digested with pure aqua ammoniæ, which will readily dissolve the sulphide of arsenic, and leave untouched most of the organic matter. The ammoniacal solution is filtered, and the filtrate carefully evaporated to dryness, at a moderate heat. If there is a sufficient quantity of arsenic present, it will have a decided yellow color; but its true nature must be established by the methods pointed out under the sulphuretted hydrogen test (see *ante*, p. 231). If, however, the dried residue contains only a minute quantity of arsenic in combination with much organic matter, a further purification of it may be necessary before the poison can be positively identified. The method of procedure will be described hereafter.

There are several other methods of examination, which will be noticed in the succeeding paragraph under the head of *Detection of the poison in the tissues.*

The *intestines and their contents* may be examined in the same manner as that above described for the stomach and its contents.

Separation from the tissues.—In all toxicological investigations, the separation of a metallic poison from the different organs of the body should always be attempted, inasmuch as this furnishes incontestable proof of the actual *absorption* of the poison from the stomach into the tissues; provided, always, the absence of post-mortem imbibition can be proved (see *ante*, p. 40). Moreover, it is this absorbed portion of the poison which has been the real cause of death, whereas that portion found in the stomach is only the residue, or complement, of that which has produced the fatal result. Besides, it frequently happens, especially when the dose of the poison has not been excessive, that all of it may have disappeared from the stomach before death, so that this organ will furnish none for chemical analysis; whilst the different organs may still contain it in notable quantity, as the result of absorption. Absorbed arsenic is readily deposited in all the soft tissues of the body, and any of these may be examined for it after death; the liver, however, usually contains the largest relative amount. The absolute quantity thus found rarely exceeds a grain in weight.

All the different methods adopted for the recovery of absorbed arsenic from the tissues have reference to one common end—the destruction of the organic matter before applying the usual tests. In some instances, however, the poison may be obtained by simply boiling the finely-divided tissue with dilute hydrochloric acid, and then employing Reinsch's method. This mode will often suffice to separate arsenic from the liver.

The following are the most approved methods of effecting the destruction or carbonization of the organic matters:

1. *By hydrochloric acid and chlorate of potash.*—This is the method of Fresenius and Babo, and commends itself by its simplicity, and by the fact that the acid employed is the same as that used in Reinsch's test. The solid organic matter (as, about one-fourth of the liver) is to be finely divided, and brought to the consistence of thick gruel, by mixture with pure water. The whole is then to be digested in a porcelain evaporating-dish, over a water-bath, with an amount of pure hydrochloric acid equal to the weight of the dry solid matter present. About twenty grains of powdered chlorate of potash are then added to the hot liquid, and the addition is occasionally repeated, stirring the liquid, until the organic matters are completely disintegrated. During this process, a little water is added to prevent concentration of the mixture. When the liquid has cooled, it is transferred to a muslin strainer, the solid residue washed repeatedly with warm water, and the washings collected separately; they are now concentrated in a water-bath, and, after cooling, added to the original filtrate: the mixed liquids are then filtered through paper. Any arsenic present would now exist in the liquid as *arsenic acid.*

The liquid is next poured into a flask, and a few drops of a strong solution of sulphite of soda are added, until it smells strongly of sulphurous acid. The object of this is to deoxidize the arsenic acid, and convert it into arsenious acid, which latter condition experience has proved to be more favorable for precipitation by means of sulphuretted hydrogen. The flask is then heated in a water-bath until all the odor has disappeared; it is then allowed to stand in a cool place for several hours, and any deposit that forms is removed by filtration. The resulting solution may now be examined for arsenic, by the different methods already described.

A given portion is treated with sulphuretted hydrogen, with all the precautions given on p. 232; and when the *quantity* of the poison discovered is to be determined, the most scrupulous care is to be observed. The precipitated sulphide should be washed on the filter, at first with water containing a little sulphuretted hydrogen, and until the washings no longer contain chlorine.

This precipitated sulphide, it is to be remembered, contains organic matter and sulphur. It will also be mixed with the sulphides of other metals that may happen to be present (*antimony*, *mercury*, *lead*, and *copper*). For the purification of the precipitate, several methods have been proposed. The one originally described by Otto, and recommended by Wormley, is highly satisfactory. The filter containing the moist precipitate is to be spread out on a porcelain dish, stirred first with distilled water to the consistence of a paste, then with excess of pure aqua ammoniæ. Any sulphuret of arsenic present, along with more or less of organic matter, will be dissolved, while the other sulphides above mentioned would remain unchanged. The mixture is now transferred to a small moistened filter, and the solid residue washed from the spritz-bottle with diluted ammonia. The filter with its contents should be reserved for future examination, if necessary.

The ammoniacal filtrate, which has usually a dark-brown color, is now evaporated, in a small porcelain capsule, to dryness, on a water-bath. It still contains organic matter. A small portion of concentrated nitric acid is next added, and the mixture again evaporated to dryness, the addition of nitric acid being repeated until the moist residue has a yellow color. The residue is then moistened with a few drops of a concentrated solution of caustic soda, a small quantity of pure carbonate and nitrate of soda added, and the well-stirred mass cautiously evaporated to dryness; the heat is then very gradually increased until the mass becomes colorless, when the organic matter may be considered entirely destroyed. In the performance of this operation, it is of the utmost importance that the nitric acid and the soda compounds employed be perfectly free from chlorine, the presence of which might cause a portion, or the whole, of the arsenic to be volatilized as a chloride.

In the incinerated residue thus obtained, the arsenic would exist in the form of the *arsenate of soda*, mixed with sulphate, nitrate, nitrite, and carbonate of soda. When cool, this mixture is dissolved in warm water, and the solution, after filtration, if necessary, strongly acidulated with pure sulphuric acid, and then evaporated until dense white fumes appear. By this treatment the carbonic, nitrous, and nitric acids are entirely expelled, leaving only the arsenate and sulphate of soda.

A portion of the strongly acid liquid thus obtained may now be tested by Marsh's process, as before described (see p. 232). But if it is desired to apply the other tests, it will be requisite to reduce the arsenic acid to the state of arsenious acid, by the use of sulphurous acid, or the sulphite of soda, in the manner directed at page 246. If the object be to ascertain the amount of the poison present, a given quantity of the acid liquid is to be treated with sulphuretted hydrogen gas, as already described, and the precipitated sulphide, after thorough washing, dissolved in aqua ammoniæ, evaporated carefully to dryness, and carefully weighed. Every 100 parts of the dried tersulphide correspond to 80.48 parts of arsenious acid. A portion of the dried sulphide, when heated in a reduction-tube, should completely volatilize without charring, or leaving any residue; otherwise, it is not perfectly pure.

A preferable method of estimating the quantity of arsenious acid present in an organic liquid, according to Wormley (Micro-Chem. of Poisons, p. 310), is to introduce a given measure of the above organic liquid into an active Marsh's apparatus, so arranged as to evolve a very slow stream of gas, and conduct the gas into a properly diluted solution of nitrate of silver. As already explained, the whole of the arsenic will be recovered as arsenious acid in solution, while the silver will be precipitated in the metallic form. When the evolved gas ceases to yield any further precipitate, the solution is filtered, and any undecomposed nitrate of silver is precipitated by the cautious addition of hydrochloric acid, as chloride of silver: this is separated on a moist filter; the filtrate is treated with sulphuretted hydrogen, in the usual manner, and the precipitate dried and weighed.

2. *Carbonization by sulphuric acid.—Method of Danger and Flandin.*—The organic tissue is cut up into fragments, and mingled with the liquid matters previously concentrated on a water-bath, and the whole introduced into a glass tubulated retort, which is attached by means of an adopter to a properly-cooled receiver. Strong sulphuric acid is then poured upon the mass, in the proportion of one-fourth its weight. The retort, which should be about one-third full, is now to be carefully heated on a sand-bath, until the black, pasty mass first produced becomes dry and carbonaceous. White fumes of sulphuric acid, highly offensive, are copiously disengaged. After cooling, the black mass is removed from the retort, first carefully breaking it up by means of a glass rod, and is reduced to powder in a glass mortar. This powder is moistened in a porcelain capsule with about one-tenth its weight of pure concentrated nitric acid, and exposed to the heat of a water-bath for half an hour. By this time all the arsenic will have been converted into arsenic acid. Warm distilled water is now added, and the whole thrown upon a filter. If the carbonization is complete, the filtrate is colorless; if it still retains a yellowish tint, it must be evaporated to dryness, first, however, adding a small quantity of sulphuric acid; the dry residue is treated with pure nitric acid, water again added, and filtration performed a second time. The carbon is washed upon the filter by successive applications of hot distilled water, and the filtrates mixed with the liquid which first passed through. This liquor is very acid, and contains a considerable quantity of sulphuric and nitric acids; it is next evaporated, first on a water-bath, afterwards on a sand-bath, until the nitrous odor has entirely disappeared. It is now to be mixed with its volume of distilled water, and filtered, if any deposit of sulphate of lime takes place. The liquid is then in the proper condition to be subjected to Marsh's test. Or, the solution may be saturated with sulphurous acid gas to convert the arsenic acid into arsenious acid, then heated to expel the excess of the gas, and treated with sulphuretted hydrogen. Or, again, it may be examined by the process of Reinsch.

The distillate, resulting from heating the original material in the retort, may contain some of the arsenic in the state of *chloride:* it should, therefore, be examined for this compound.

3. From the fact that when arsenious acid is treated with hydrochloric acid it is converted into the terchloride, advantage may be taken to separate this poison from the tissues, or from any organic mixture, by this means. It is necessary completely to dry the tissue, first cut into pieces, by evaporation on a water-bath; then, after mixing it with about its own weight of concentrated hydrochloric acid, to distill it on a sand-bath to almost dryness,—the distillate being collected in a well-cooled receiver. The residue in the retort may be redistilled with a fresh portion of the acid.

The distillate thus obtained contains the arsenic as *terchloride*, together with free hydrochloric acid, and some organic matter. A portion of the distillate may be examined by Reinsch's test; other portions, properly diluted, may be tested with sulphuretted hydrogen, and by Marsh's method.

Should the arsenic have existed in the substance distilled as a sulphide (as sometimes happens in the body many months after death), it will not appear in the distillate. Under such circumstances, the residue in the retort may be treated with diluted hydrochloric acid, and the occasional addition of chlorate of potassa, until the organic matter is destroyed: the resulting solution, after being treated with sulphurous acid, may be examined as above.

The urine.—This liquid may be concentrated by heat to a small volume, strongly acidulated with hydrochloric acid, and examined by Reinsch's process. Or, it may be evaporated to dryness on a water-bath, the residue treated with hydrochloric acid and chlorate of potassa, in the usual way, and then with sulphurous acid gas: the resulting liquid can be employed for the usual tests.

The urine appears to be the principal secretion through which arsenic is eliminated from the system. It has been detected in the urine as late as the twenty-first day after being taken.

The following *résumé* of facts bearing practically on the detection of arsenic in the human body is taken from Guy's Forensic Medicine, p. 444:

"Arsenic may be detected in the dead body after such long intervals of time as five, seven, ten, or even fourteen years.

"Arsenious acid, usually found attached to the coats of the stomach as a white powder, or paste, is converted into the yellow sulphide by the sulphuretted hydrogen generated by putrefaction.

"Preparations of arsenic preserve dead animal matter.

"Orfila affirmed that arsenic is a natural constituent of the body itself; and that it may be discovered both in the fleshy parts of the body, and in the bones. But subsequent researches of himself and others have shown that there was in his first experiments some source of fallacy.

"Arsenic, when contained in the soil of cemeteries, is generally, if not always, in an *insoluble* form, in combination with iron or lime.

"Preparations of arsenic, whether taken in single large doses, or in repeated small ones, are absorbed into the blood, and may be found in the textures and secretions; and they are only slowly eliminated from the body. The limit usually stated for the complete elimination of arsenic from the human body is three weeks; but it has been extended to a month by M. Bonjean (Ranking's Half-Yearly Abstract, vol. iii.)."

OTHER PREPARATIONS OF ARSENIC.

Arsenite of potassa.—This is the active principle of *Fowler's solution*, a form in which arsenic is much used in medicine. It contains four grains of arsenious acid to the fluidounce of the liquid. It may be readily tested by any of the methods described for arsenious acid.

Arsenic acid.—This acid, though a powerful poison, is of no medico-legal interest except as being formed in some processes for detecting arsenious acid. It is a white, deliquescent solid, very soluble in water, not completely volatilized by heat, and having a strong acid reaction. It yields a metallic sublimate when reduced by charcoal, and a metallic deposit on copper when treated by Reinsch's process; also a metallic crust by Marsh's test. Sulphuretted hydrogen produces in it, after a time, a yellow precipitate; it is hastened by boiling.

Its most delicate test is *nitrate of silver* in solution, which gives a brownish-red precipitate—arsenate of silver.

Arsenite of copper (Scheele's green).—This is a fine green powder, consisting of one part of arsenious acid and two of oxide of copper. It yields distinct crystals of arsenious acid when heated in a reduction-tube, and a residue of oxide of copper. It is soluble both in ammonia and in nitric acid.

Aceto-arsenite of copper (Schweinfurt or Brunswick green, Vienna green, Emerald green).—This pigment is largely used for coloring wall-papers; also to give color to confectionery, toys, bonbons, etc., and to articles of dress and millinery. It is composed of arsenious acid six parts, oxide of copper two parts, and acetic acid one part. It is readily identified by heating it in a test-tube, when it gives off strong fumes of acetic acid, depositing crystals of arsenious acid, and leaving a residue of oxide of copper.

Paper or other material colored with this pigment will readily indicate the poison by being dipped in a weak solution of aqua ammoniæ: the material will be bleached, while the solution becomes blue; if now a crystal of nitrate of silver is dropped into it, it instantly turns yellow around its edges, from the formation of the arsenite of silver. Green wall-paper colored with this pigment may at once be tested by applying to it a drop of aqua ammoniæ, which will produce a deep-blue spot.

As already mentioned, poisoning frequently occurs from inhaling the fine dust or powder detached from walls covered with this sort of green or yellow paper. Persons employed in manufacturing artificial leaves, fruits, and flowers—chiefly young women—suffer frequently from this sort of chronic poisoning, which may sometimes proceed even to a fatal issue.

Among the effects produced upon those who work in materials containing arsenite of copper, a peculiar papular eruption, running into pustulation, is one of the most common. It appears about the nostrils, back of the ears, on the bends of the elbows, the inside of the thighs, and then the scrotum.

Occasionally the fingers are affected, when the nails are apt to drop off. These symptoms disappear on abandoning the employment.

Sulphides of arsenic.—There are two *native* sulphides,—the *tersulphide*, or orpiment, and the *pentasulphide*, or realgar. The artificial sulphides are named orpiment, or *yellow arsenic*, and realgar, or *red arsenic.* Both of the latter are employed in the arts, as colors. The pigment called *king's yellow*, as well as the orpiment, contains arsenious acid in considerable quantities: they are both highly poisonous.

The yellow sulphide is occasionally taken as a poison; and it is often found adhering to the coats of the stomach and bowels after death, having been formed, as before stated, by the union of arsenious acid with the sulphuretted hydrogen generated by decomposition. In organic mixtures, the sulphides are detected by their characteristic colors: they are soluble in ammonia, and from this solution they are precipitated by hydrochloric acid.

The sulphides, when mixed with black flux or ferrocyanide of potassium, and heated, yield metallic sublimates (see p. 232).

The symptoms, post-mortem appearances (except the yellow color), and treatment, in cases of poisoning from the yellow sulphide, are similar to those already detailed under the head of Arsenious Acid.

CHAPTER XV.

POISONING BY ANTIMONY.

UNTIL within comparatively few years, Antimony has not figured largely among poisons. But latterly, cases of slow poisoning by tartar emetic have become quite frequent, especially in England.

The only preparations of Antimony of medico-legal interest are Tartar Emetic and the Chloride.

TARTAR EMETIC (*Tartarized Antimony.—Stibiated Tartar.—Tartrate of Antimony and Potassa*).—*Properties.*—This is a double salt, composed of tartaric acid in combination with teroxide of antimony and protoxide of potassium. It occurs, when pure, in large, colorless, octahedral crystals, or as a white powder. As found in commerce, it occasionally contains traces of arsenic. When heated in a reduction-tube, over the flame of a spirit-lamp, it readily blackens, from the decomposition of its organic acid, and is soon reduced to a mixture of carbon and metallic antimony. Heated on charcoal before the blowpipe, it is also reduced, yielding globules of the metal, which, when cooled, are very brittle, and at the same time an abundant white incrustation of the oxide.

It is soluble in about three parts of boiling, and fifteen of cold, water. Its solution speedily undergoes decomposition, the organic acid appearing to develop a filamentous growth. It is insoluble in alcohol, this liquid precipitating it from its aqueous solution. From a strong solution in water, it separates on evaporation in well-defined tetrahedral crystals, or some modification of the cube. Often, both varieties are found in the same specimen. If the solution is very weak, the deposit is apt to be undefined. The best crystals are procured from hot solutions on cooling.

The *taste* is generally described as nauseous, acrid, metallic, or austere. On some palates, however, it seems to produce very little, if any, impression. Dr. George B. Wood (Therapeutics, vol. ii. p. 58) describes the taste as slightly sweetish and somewhat styptic.

Symptoms.—A large dose of tartar emetic is usually followed by the following symptoms: a styptic, metallic taste perceived on swallowing; this is shortly succeeded by nausea, retching, violent and incessant vomiting, a sense of heat in the throat, with difficulty of swallowing, great thirst, burning pain in the stomach and bowels, profuse purging of a watery character; the dejections from the bowels, as well as the matters vomited, sometimes contain blood, and often bile. There are severe cramps in the extremities; a small, frequent pulse; great prostration; a tendency to syncope; a cold skin (sometimes, according to Orfila, an intense heat of skin); and a clammy perspiration. The urine is generally increased in quantity, and is sometimes voided with pain. According to Tardieu (Sur l'Empoisonnement, p. 608), the urine is scanty. Towards the close of fatal cases, it is apt to be scanty and bloody, and even suppressed. Should the case terminate fatally, convulsions and delirium may, in exceptional cases, precede death, just as sometimes occurs in arsenical poisoning. Large doses occasionally produce insensibility, as one of their earliest effects; and in some rare instances, after a very large quantity has been swallowed, there has been a total absence of vomiting and purging until emetics were administered. (See a remarkable case reported by Dr. Gleaves, in Western Jour. of Med. and Surg., Jan. 1848.) In such cases, according to Orfila (Toxicologie, 1852, vol. i. p. 626), the intensity of the other symptoms is increased. Husemann (Toxicologie, p. 853) speaks of this *exceptional* form of antimonial poisoning in which there is an absence of both vomiting and purging; the symptoms being intense prostration, cold, clammy sweat, a sense of oppression in the chest, the respiration at first increased, then diminished in frequency, and embarrassed; a rapid, feeble pulse, afterwards becoming slow, intermittent, and irregular; delirium, unconsciousness, tremblings; and clonic and tonic convulsions.

Two cases are recorded by Dr. John Elliotson (Med. Times and Gaz., July, 1856) in which *convulsions* took place. These were infants, aged respectively fourteen and eight months, and suffering from inflammatory croup. To the former, tartar emetic was administered for four consecutive days, in half-grain doses, every few hours, until *twenty-seven grains* were taken in the aggregate. The most dangerous prostration succeeded the violent vomiting and purging, and finally convulsions ensued, in which there was considerable rigidity of the limbs and jaws. The second case was also treated with half-grain doses of tartar emetic every hour, until *seven* grains had been given in the course of sixteen hours. In this case likewise, after great prostration, there were convulsions of a rigid character. Both children ultimately recovered. We think it extremely doubtful whether the convulsions in these cases were primarily due to antimony: it is well known that such convulsions are apt to ensue in cases of rapid exhaustion from any cause, as from hemorrhage, or serous diarrhœa.

Another occasional symptom, if the patient survives the third or fourth day, is the appearance over the body of the true pustular eruption which characterizes the external application of tartar emetic.

Most frequently, a single large dose does not prove fatal; the prompt and abundant vomiting of the poison preventing its absorption.

The fact of the *tolerance* of large doses of tartar emetic in certain diseased states of the human system is sufficiently established. Rasori, Laennec, and others have given it, in pneumonia, acute bronchitis, and acute rheumatism, in enormous quantities, without producing the usual poisonous symptoms. This *contra-stimulant plan*, as it was named, consisted in administering the remedy in rapidly-increasing doses every few hours, so that often as much as one or two drachms were taken in the course of a day. If the first dose or two excited nausea and vomiting, the succeeding increased doses appeared to allay these symptoms, so that the system seemed speedily to become accustomed to it.

From the numerous experiments on man and the lower animals, there can be no doubt that while antimony (tartar emetic) exerts a direct irritant impression on the gastro-enteric mucous membrane, it produces an equally direct depressant effect on the heart; so that, physiologically, it might very properly be classed among the *cardiac depressants.*

Fatal dose.—The quantity actually required to destroy life is unknown. Several instances are recorded in which very small doses produced most violent and even fatal effects; but these are probably to be regarded as exceptional cases, and as due rather to some idiosyncrasy of the patient. Thus, Dr. A. Stillé (Ther. and Mat. Med., ii. 346) relates the case of an insane female, of general good health, in whom a dose of not more than half a grain occasioned violent vomiting and purging, and a state closely resembling the collapse of cholera. Dr. Taylor (On Poisons, p. 389) records a case in which four grains very nearly proved fatal. Vomiting, purging, and convulsions took place, followed by collapse, with failure of the heart's action and coldness of the skin. Of thirty-seven cases of tartar emetic poisoning collected by Dr. Taylor, sixteen proved fatal. Of the fatal cases, the smallest dose was in a child, *three-quarters of a grain;* and in an adult, *two grains;* but in the latter case the patient had been in a previous enfeebled state of health. (Guy's Hospital Reports, Oct., 1857.)

Dr. C. A. Lee (N. Y. Med. and Phys. Jour., No. xxx. p. 302) reports the case of a child a few weeks old, in which a dose of *fifteen grains* proved fatal. Dr. Récamier (Orfila, Toxicol., 1852, i. p. 623) details a case of a healthy man, aged fifty years, who died within four days, after taking rather less than *forty grains.* Probably, twenty to forty grains may be regarded as the usual minimum fatal dose for an adult.

On the other hand, numerous instances are recorded of recovery after swallowing enormous doses—varying from one drachm to an ounce. The last-named quantity was taken in the case reported by Dr. Gleaves, already alluded to. Vomiting was only excited by artificial means an hour and a half after the poison was swallowed. The man recovered perfectly in about two weeks. In a case related by Dr. McCreery, U. S. N., a physician swallowed half an ounce of tartar emetic, by mistake for Rochelle salts. In about half an hour, nausea, vomiting, and purging set in, followed by violent cramps of the legs and arms. He took freely of infusion of green tea and tannin, with other remedies. Though very much prostrated, he recovered completely in a few days. (Am. Jour. of Med. Sci., Jan., 1853.)

Fatal period.—Prof. Wormley (Micro-Chemistry of Poisons, p. 218) relates the case of a child recovering from measles, who died in an hour from the depressing effects of three-quarters of a grain of tartar emetic prescribed as a medicine. The same authority quotes a case of Dr. C. Ellis (Boston Med. and Surg. Jour., Dec., 1856), in which an unknown quantity of the poison proved fatal to a young lady, aged twenty-one years, *in seven hours.* Dr. Pollock reports a case (Lon. Med. Gazette, May, 1850) in which sixty grains proved fatal to a robust, healthy man in the course of ten hours.

In the greater number of instances, death does not take place so rapidly, several days usually elapsing before the fatal issue. A case is related by Deutsch (Canstatt's Jahresbericht für 1851, Bd. iv. p. 270), in which a woman took by mistake a scruple of tartar emetic: she suffered much from its violent action, and died in a year, from the irritant effects on the intestinal canal.

Post-mortem appearances. — M. Tardieu very correctly remarks (*loc. cit.*, p. 611) that the examination of the body in a case of death from tartar emetic poisoning does not always furnish positive data. It often happens that we can discover no appreciable lesion. This was well illustrated in the case of the two women poisoned by Dr. Pritchard, as detailed by M. Félizet (Archives Gén. de Méd., Sept., 1865), who remarks that the chief matter of scientific interest in the case is that it affords an example of poisoning by tartar emetic, without leaving any anatomical lesions. This observation, however, has reference rather to a case of slow poisoning, in which the tartar emetic was administered in small and repeated doses.

According to Tardieu (*loc. cit.*), most commonly, and especially when a single very large dose has been taken, tartar emetic produces numerous and extensive lesions. The œsophagus is reddened and sometimes ulcerated. The stomach and intestines are extensively inflamed, as shown by a deep redness, with softening, of the mucous membrane, upon which are scattered brownish-red or blackish patches, formed by the infiltration of blood and by hypertrophy of the follicles. The inner surface of the stomach and small intestines is covered with a blackish, thick, and viscid secretion, sometimes streaked with blood. Occasionally there may be found in the primæ viæ, and even in the commencement of the small intestines, true pustules filled with pus.

The *liver* is generally enlarged, and appears to have undergone a fatty degeneration. In confirmation of this pathological change, it is stated, as a well-authenticated fact, that the peasants of the duchy of Brunswick have long been accustomed to feed their geese upon the white oxide of antimony, in order to fatten them. The *lungs* are often deeply congested: some cases exhibit a true apoplexy; and the interior of the *bronchi* and *trachea* is uniformly reddened. The *brain* is usually more or less congested, both in its membranes and in its substance: the latter presenting, when cut, numerous bloody points. Sometimes there is a slight serous infiltration at the side of the brain. The *heart* exhibits nothing abnormal. The *blood* is less coagulated than in health.

In Dr. Lee's case, before mentioned, the mucous membrane of the stomach was red and softened. The stomach contained a small quantity of slimy mucus. The duodenum was of a deep-brown color, almost livid, and contained the same sort of viscid matter as the stomach. The inflammation did not extend into the large intestines. The vessels of the scalp, as well as those of the brain, were distended with blood. The ventricles were half filled with serum; and there was also effusion beneath the pia mater.

A series of experiments by Dr. Nevins, detailed by Prof. Guy (Forensic Medicine, p. 460), exhibits the effects produced upon rabbits by small and repeated doses of tartar emetic. The symptoms were loss of appetite, loss of spirit, and emaciation. None of them vomited; diarrhœa occurred in three out of eight. There were no cramps; convulsions occurred in four, just before death. Out of the eleven experimented upon, five died, at intervals of four to seventeen days after commencing to take the poison.

The post-mortem lesions were *congestion of the liver* in all; *redness of the mucous membrane* of the stomach in most of them; *ulceration* in two; the *small intestines* showed patches of inflammation throughout in some; the *solitary glands* were enlarged in two; they were of a yellow color, and loaded with antimony; the *cæcum* and *rectum* were nearly always healthy; the *lungs* were generally deeply congested; in some actually inflamed and hepatized; the *air-tubes* were of a bright-red color; the *brain*, *heart*, and *spleen* were healthy; the *kidneys* were more or less congested. Bloody exudations were sometimes found in the cavities of the chest and abdomen.

The poison was discovered in every part of the body,—always in abundance in the liver, in less quantity in the spleen; earliest in the tissues of the stomach; later in the kidneys and cæcum; at an early period in the lungs. It was difficult to detect it in the muscles and in the blood, but it was found in the bones as late as the thirty-first day after discontinuing the poison. The poison was being constantly eliminated by the kidneys, commencing very early, and continuing for twenty-one days after it had been suspended.

Treatment.—Vomiting should be assisted by warm mucilaginous drinks, and, if necessary, by tickling the throat with a feather; or the stomach-pump may be employed. The proper *antidote* is tannin in some form, as tincture or infusion of cinchona, and infusion of green tea, or of galls. After evacuation of the stomach, opium may be given with advantage. Stimulants, both internally and externally, are demanded. Any resulting inflammations must be treated on ordinary principles.

Chronic, or slow poisoning.—Many cases have recently come to light, in which death has undoubtedly been occasioned by small and repeated doses of tartar emetic. The symptoms are nausea, uneasiness, retching, occasional vomiting, diarrhœa, with pasty stools; the abdomen painful and distended; loss of appetite, and emaciation; the tongue is slimy, and the mouth clammy; the head feels full and heavy. Later, there is slowness and loss of power in the heart's action; the breathing is difficult; the complexion is dusky; there is stiffness of the legs, with great debility. The countenance is pale and anxious; there is a disposition to sleep, with inability to maintain the erect position; faintings; flushing of the face; increase of perspiration, and of the urinary secretion.

External application.—Tartar emetic not only occasions *pustulation* when applied to the skin, but it is capable also of producing its general impression on the system by absorption from this surface. Even fatal consequences have resulted from its application to abraded surfaces. Tardieu (Sur l'Empoisonnement, p. 610) mentions the case of a woman who was induced by a quack to apply to an open sore on her breast a salve composed of equal parts of tartar emetic and lard. The unfortunate patient died some days after, with all the symptoms of violent poisoning.

Chemical analysis.—(1) *As a solid.*—When moistened with a solution of sulphuretted hydrogen, or with sulphide of ammonium, it immediately acquires an orange-red color. This is characteristic of a salt of antimony *in its pure state.* The action of heat upon tartar emetic in the solid form has already been described (p. 254).

(2) *As a liquid.*—(*a*) On slowly evaporating a drop of the solution on a piece of glass, it will crystallize in tetrahedra, or some other modification of the cube. If the solution be very dilute, the crystallization is confused. (*b*) Either nitric, hydrochloric, or sulphuric acid, when diluted and dropped into the solution, throws down a white precipitate, soluble in an excess of the acid. Nitric acid is preferable in this experiment, inasmuch as the other two acids precipitate numerous other metallic substances. This white precipitate is easily soluble in *tartaric acid.* (*c*) *Ferrocyanide of potassium* does not precipitate the solution,—whereby tartar emetic is distinguished from most other metallic poisons. (*d*) When acidulated with hydrochloric acid (one-sixth part), and boiled in contact with bright *copper*-foil, the latter becomes speedily incrusted with a violet or gray-colored coating of *metallic* antimony. (*e*) The solution, containing one-tenth part of hydrochloric acid, gives a black coating to a piece of pure *tin*-foil, *in the cold*,—whereby it is distinguished from arsenic. (*f*) *Sulphuretted hydrogen*, or *sulphide of ammonium*, throws down, from a *pure* solution, the characteristic orange-red tersulphide of antimony. This action is facilitated by acidulating the solution with hydrochloric acid. This precipitate is soluble in the fixed caustic alkalies, but almost entirely insoluble in ammonia (in which respects it differs from sulphide of arsenic). It is not soluble in dilute hydrochloric acid; but the hot concentrated acid readily dissolves it with the escape of sulphuretted hydrogen gas. The resulting solution (terchloride), if not too acid, when dropped into a large quantity of water, immediately throws down a copious, flaky, white precipitate (the oxychloride, or powder of Algaroth): this is quite characteristic of antimony, and the precipitate may be identified as *antimonial* (1) by its complete solubility in tartaric acid, and (2) by touching it with sulphide of ammonium, which will immediately impart to it an orange-red color. The white precipitate obtained by

treating *bismuth* as above is not soluble in tartaric acid, and is immediately *blackened* by sulphide of ammonium.

(*g*) *The galvanic test.*—This consists in putting a drop or two of the solution acidulated with hydrochloric acid into a platinum capsule, or upon a piece of platinum-foil, and touching the platinum, through the liquid, with a piece of pure zinc. Metallic antimony is very soon deposited on the platinum surface, at the point of contact, as a black or brownish film. The liquid should then be poured off, and the platinum thoroughly washed in distilled water. A small quantity of sulphide of ammonium poured upon it, speedily dissolves the deposit (if antimony) by the aid of heat; and on evaporation an orange-red sulphide remains. This may, in turn, be dissolved by a few drops of strong, hot hydrochloric acid, and the solution, on being dropped into water, will precipitate the white oxychloride (see p. 261). A modification of this galvanic test may be advantageously applied to the discovery of antimony in the organs (see *post*).

(*h*) *Marsh's test* (*antimonetted hydrogen*).—When a solution of tartar emetic, or of any of the soluble antimonial salts, is subjected to Marsh's test, metallic antimony unites with the nascent hydrogen, and *antimonetted hydrogen gas* is given off, under conditions precisely similar to those pertaining to arsenic (see p. 232). Similar precautions should be observed to those mentioned under the head of Arsenic.

1. If the gas, as it escapes from the orifice of the delivery-tube, be ignited, it burns with a bluish flame, evolving white fumes of the teroxide of antimony, unless the amount of the metal present be extremely minute. If these fumes be received in a short, wide test-tube, held just above the top of the flame, a white deposit of the *teroxide* may be collected: this deposit is not distinctly crystalline, as in the case of arsenic; when touched with sulphide of ammonium it immediately acquires an orange-red color. If a piece of cold white porcelain be held low down in the flame, the metal is deposited (as in the case of arsenic) in the form of a black (or nearly black) spot, which is usually surrounded by a grayish ring. By frequently changing the position of the porcelain, any number of these metallic deposits may be procured.

The only other metal which, under the above conditions, will yield similar spots, is arsenic. A little attention will, however, enable us to distinguish them. They usually differ in their physical appearance,—those from antimony being generally dull, resembling soot, while those from arsenic usually present a brilliant metallic lustre, and are of a steel-gray or brownish color. This distinction is not, however, invariably presented: if the antimony be in very minute proportions, and the spots proportionally small, we have seen them exhibit precisely the appearance of arsenical spots. They differ, however, greatly in other properties. Thus, the antimony deposits are slowly dissipated by the flame of a spirit-lamp, while those from arsenic are readily volatilized. Again, the antimony spots readily dissolve in sulphide of ammonium, while the arsenical are but slowly affected by it. Moreover, the antimonial solution, if evaporated to dryness, leaves an orange-red residue, which is insoluble in ammonia, but soluble in strong, hot hydrochloric acid; whilst that from arsenic yields, on evaporation, a yellow residue, which is soluble in ammonia, and insoluble in hydrochloric acid. Furthermore, the antimonial spots are insoluble (or nearly so) in a solution of hypochlorite of soda or lime, whilst the arsenical spots immediately disappear, on being moistened with the solution. Nitric acid also serves to distinguish them: both are dissolved by it; but on evaporation to dryness, the arsenical residue gives to a solution of nitrate of silver a brick-red stain (arsenate of silver), whilst the antimonial residue is not affected at all by the same reagent.

2. If heat be applied to the horizontal tube in Marsh's apparatus, through which the antimonetted hydrogen is passing, decomposition of the gas ensues, metallic antimony being deposited in the form of a dark-gray, shining ring. If the quantity of antimony experimented upon is very small, the deposit occurs wholly on the *inner* side of the part to which the flame is applied; but when in larger quantity, the deposit takes place *on both sides* of the flame. In the case of arsenic similarly treated, the mirror is formed always outside, or *in advance of* the flame.

This reaction is exceedingly delicate: according to Wormley (Micro-Chemistry of Poisons, p. 229) it will detect one ten-thousandth of a grain of the teroxide. These metallic deposits exhibit the same chemical reactions as those produced on porcelain by the ignited gas (p. 262).

3. Another method of identifying the antimonial mirror is by passing dry sulphuretted hydrogen gas through the horizontal tube, and applying the flame of a spirit-lamp to the mirror: the tersulphide of antimony is formed, which has a dark-brown, or nearly black, color. Under similar circumstances, arsenic would form the *yellow* tersulphide of this metal; but the antimonial tersulphide requires a higher heat for its production, and it is deposited much nearer the flame of the lamp than the arsenical.

4. If the antimonetted hydrogen be passed into a solution of nitrate of silver, the latter (as in the case of arsenic) becomes black; the whole of the antimony is precipitated as *antimonide of silver;* whereas, in the case of arsenic, metallic silver alone is precipitated, the arsenic remaining in solution as arsenious acid. When only a minute trace of antimony is present, the whole of the precipitate collects in the lower end of the delivery-tube, in the form of a black ring (Wormley).

It should be remembered that the mere production of a black precipitate, under these circumstances, is not sufficient to prove the presence of antimony, since sulphur, phosphorus, and other bodies might occasion similar-looking deposits. The true character of the antimonide of silver may be shown by collecting the deposit on a filter, washing with warm water, and boiling with dilute hydrochloric acid, in which the antimony will dissolve, while the silver is insoluble. After filtration, the solution is treated with sulphuretted hydrogen, which will yield the characteristic tersulphide. Or, according to Prof. Hofmann (Quar. Jour. Chem. Soc., April, 1860), the washed antimonide of silver may be boiled with a solution of tartaric acid, which readily dissolves the antimony, leaving the silver untouched; the solution is filtered and treated with sulphuretted hydrogen. By either of these methods, an exceedingly small quantity of antimony may be recognized.

An additional recommendation of this process is that it enables the analyst to detect either antimony or arsenic in the presence of each other.

In organic mixtures.—As tartar emetic is precipitated by tannic acid, but not readily by albumen or mucus, it may generally be found in the contents of the stomach partially dissolved, provided no antidote has been administered. In a case of suspected poisoning, it would be very desirable to separate the tartar emetic, *as such;* although the detection of the presence of the antimony alone is usually regarded as sufficient to establish the proof of administration, since this is really the poisonous element. This separation may be conveniently effected by the process of *dialysis*, already described (see page 113). The exhibition, at the trial, of the *identical* poison that had caused death, as extracted from the stomach of the deceased, is always very conclusive evidence with a jury. Of course, this same process may be employed to separate the poison from suspected food and drinks, or from vomited matters; but, obviously, it cannot be used for detecting the *absorbed* poison, in the tissues or secretions.

The suspected organic matters, with the addition of distilled water, if necessary, should be acidulated with tartaric acid, and exposed to a gentle heat for about half an hour. When cold, the liquid should be strained through muslin, which is to be washed with distilled water and pressed, the washings added to the filtrate, and the whole carefully evaporated to about one-half. Trial tests may now be made with a portion of this liquid, by adding one-tenth of its bulk of hydrochloric acid, and inserting a piece of pure tin-foil, in the cold: the tin will receive a black coating, if antimony be present. On boiling another acidulated portion, and, while boiling, introducing a piece of bright copper, the presence of antimony will be indicated, as explained above (page 261). The remainder of the liquid should next be treated with washed sulphuretted hydrogen gas, which may be allowed to pass through it slowly for several hours, or as long as any precipitate is formed. It should then be permitted to stand in a moderately warm place for several hours, to enable the precipitate to subside. If antimony is present in comparatively large quantity, the precipitate will have more or less of an orange-red, or brownish-red, color. The pure, *bright* orange red is seen only in the absence of all organic matter.

When organic matters are present (as they must necessarily be in such mixtures), the passing of sulphuretted hydrogen for a length of time through these complex mixtures must cause a precipitate that will be composed largely of sulphur and organic matter. There can be no doubt that in some peculiar complex organic mixtures sulphuretted hydrogen—especially if passed through them for many hours—will determine a reddish, or reddish-brown, precipitate, which might suggest the suspicion of antimony, even though this poison were not present. Such a deposit would, of course, be composed merely of organic matter and sulphur, and it should be carefully distinguished from a *metallic sulphide.*

We have the authority of Tardieu for saying that, even after the precipitation of the whole of the metal from an acidulated metallic solution mixed with organic matters, by sulphuretted hydrogen, if this gas be again passed through the filtered liquid, it will throw down another colored precipitate.

Certainly, then, in the search for antimony, in a case of suspected poisoning, the merely getting a reddish, or even *orange*-reddish, precipitate by sulphuretted hydrogen, must not be deemed conclusive; neither will it suffice further to dissolve this precipitate in strong boiling hydrochloric acid, and to throw the solution into water in order to obtain the white, flaky precipitate, supposed to be so characteristic of antimony, since, as we have frequently observed, just such results may be obtained by a similar treatment of the colored sulphur-organic deposits above alluded to: these likewise are, to a great extent, soluble in the boiling acid; and the resulting solution, if allowed to fall into water, occasions a white precipitate. Now, although this sulphur-organic precipitate may not possess *all* the characters of the real sulphide of antimony—such as would result from a pure solution—it does resemble very closely, in most of its reactions, just such a preparation as we should expect to find in a *mixture of antimony and organic matters.* It is for this reason that we insist emphatically on the insufficiency of this one exclusive line of testing (viz., the sulphuretted hydrogen method) to establish the proof of the presence of antimony, in a case of alleged criminal poisoning with that substance: the most it can do is to furnish an *indication* of it; but an indication is quite a different thing from a *proof*. We do not wish to be understood as undervaluing this important test. *In pure solutions, and in the absence of all organic matters*, we regard the sulphuretted hydrogen test, followed up as detailed above, as one of the most positive and valuable tests that we possess; but only with these reservations.

In cases of poisoning involving the question of life and death, nothing but the most absolute proof of the detection of the poison by the chemist should be admitted. The analyst may, perchance, relying upon a very long experience, suppose that, while "he has satisfied himself" by this partial and imperfect method of testing, others ought equally to be satisfied. Surely the natural advance made in chemical science must detect and expose such a fallacy. This very question constituted a capital point in the defense, at the celebrated trial of Mrs. E. G. Wharton for the alleged poisoning of General Ketchum, at Annapolis, Md., in 1872. The chemist employed to make the analysis of the stomach confined himself exclusively to the above single line of testing; and by an extraordinary process of induction—comparing, *with his eye alone*, the bulk of the complex precipitate thus obtained from the stomach of the deceased, with the bulk of another substance, alleged to be sulphide of antimony, and similarly obtained from another complex organic material not connected with the deceased—he was led to *infer* the presence of twenty grains of tartar emetic in the stomach of General Ketchum!

Prof. A. S. Taylor (Prin. and Prac. of Med. Juris., 1873, p. 311), alluding to this trial, says: "The symptoms, taken as a whole, bore no resemblance to those observed in poisoning with antimony, and, but for the alleged discovery of twenty grains of tartar emetic in the stomach after death, no suspicion of poisoning would probably have arisen." "On examining the chemical evidence, it appears that the process by sulphuretted hydrogen alone was employed, and a red-brown sulphide, resembling that of antimony in chemical properties, was obtained. . . . No separation of antimony in the metallic state was made to corroborate the inference drawn from the precipitate produced by sulphuretted hydrogen. No chemical results were produced in court; though twenty grains would have allowed of the production of metallic antimony in a few minutes by copper, tin, zinc, and platinum, and by Marsh's process. The evidence that antimony was really there was not satisfactory; and that twenty grains were present in the stomach was wholly unproved. . . . The jury, upon such weak evidence, properly acquitted the prisoner."

It may be here remarked that, in a case of suspected antimonial poisoning, the production of the *metal* should be rigorously insisted upon, as the only absolute and unequivocal proof; and this, too, in quantities sufficient to admit of its positive identification by *all* the recognized tests. This proof is always required in cases of poisoning by other metallic substances, as, for example, arsenic, mercury, lead, copper, and zinc; and certainly there is no reason why it should be regarded as of less consequence in the case of antimony, especially when it is remembered with what facility this extraction of the metal may be accomplished, viz., by tin-foil, by copper, by the galvanic test, by Marsh's process (three modifications), and by the blowpipe. Dr. Taylor, in further commenting upon this subject (*loc. cit.*, p. 314), remarks: "Antimony in the metallic state is so easily procured from a small quantity of material by one or the other of the above-mentioned processes, that on no account should this be omitted. The procuring of the metal may be made subsidiary to the procuring of the sulphide, as the metal can be easily oxidized and converted into sulphide in a pure form, and obtained entirely free from organic matter. A reliance on a small quantity of a colored precipitate from sulphuretted hydrogen alone would be most unsatisfactory as chemical evidence."

Orfila is very decided upon this point: he remarks (Toxicologie, 1852, vol. i. p. 636): "What difference does it make whether the antimonial preparation occurs in soluble or in insoluble matters? The process should always be completed by the separation of the *metallic antimony;* and when this is obtained, it can easily be converted into the sulphide." Again, when remarking on the impossibility of procuring tartar emetic as such, after absorption, from the organs, and more especially from the liver, he says (p. 637), "we can obtain *the metal* from at least a portion of what has been absorbed." Once more (p. 638): "The extraction of *metallic antimony* from the viscera, or the urine, of persons who had not been using any antimonial preparation medicinally, is an incontestable proof of poisoning."

Tardieu is equally emphatic on the necessity of obtaining the *metallic* proof of antimony, in medico-legal researches. He says (Sur l'Empoisonnement, p. 619), after mentioning the two methods of getting rid of the organic matters, viz., by carbonization with sulphuric acid, and by hydrochloric acid and chlorate of potassa, "Finally, we complete the indications obtained from the residue by introducing a portion of the liquid into a Marsh's apparatus" (of course with the view of obtaining the *metal*).

When it is desired to ascertain the precise quantity of the poison found either in organic mixtures, or in the tissues of the body, the crude sulphide, procured in the manner above indicated from a given portion of the material, must first be brought to a state of absolute purity (see *ante*, ARSENIC, p. 247), and then, after thorough drying, be accurately weighed. Every 100 parts by weight of the pure tersulphide of antimony correspond to 85.74 of the teroxide, or 202.85 parts of crystallized tartar emetic.

The purification of the sulphide may be conveniently effected (Wormley, Micro-Chemistry of Poisons, p. 234) by washing the precipitate and transferring it to a thin porcelain dish, treating it with a few drops of concentrated nitric acid, and cautiously evaporating it to dryness; the operation being repeated, if necessary, until the organic matter is well carbonized. Any antimony present will now exist as *antimonic acid.* The residue is then moistened with a few drops of a strong solution of potassa, evaporated to dryness at a moderate heat, and the dry residue very gradually heated to fusion. The cooled mass is stirred with a little water, the mixture acidulated with tartaric acid, then boiled for some minutes, and the solution filtered. The whole of the antimony will now be present in the filtrate, which, if the operations have been conducted with care, will be perfectly colorless. This solution, slightly acidified with pure hydrochloric acid, is to be treated with sulphuretted hydrogen, as already described (p. 247). The product is the pure sulphide, which, when properly dried and weighed, will afford the data required.

The above authority states that by this method the sulphide of antimony produced from the one-hundredth of a grain of the teroxide of the metal may be recovered from a very complex organic mixture, without any apparent loss. Should the final tartaric acid solution prove to be colored, then, instead of treating it with sulphuretted hydrogen, it may be put into a Marsh's apparatus, and the resulting antimonetted hydrogen be received into a solution of nitrate of silver; the washed black precipitate of antimonide of silver is boiled with tartaric acid, and the solution filtered, and treated with sulphuretted hydrogen, as above.

In the tissues.—As already mentioned, antimony, when swallowed into the stomach, speedily passes into the circulation, whence it is soon deposited in the various tissues and organs of the body. Of these, the liver and kidneys generally contain the largest amount of the absorbed poison. A weighed portion of the liver (about one-quarter) should be cut up into fine pieces, and boiled in pure water acidulated with one-sixth of hydrochloric acid. After some time, a trial test may be made with a slip of pure copper, allowing sufficient time for any deposit to take place. This deposit should be verified in the manner pointed out on page 240. On heating one or more of these dried copper slips in a clean, dry reduction-tube, there will be a white sublimate deposited in the cool portion of the tube. This is not composed of octahedral crystals, as is the case with arsenic (see ARSENIC, *Reinsch's test*, p. 238), but will be found to be either granular, or composed of very fine acicular crystals (Millar's Inorganic Chemistry, p. 602). Mercury, under similar circumstances, is deposited in minute metallic globules, easily recognized by means of a magnifier.

The true nature of this antimonial deposit may be shown, as first advised by Watson, by boiling the coated copper in a dilute solution of caustic potassa, the metal being occasionally withdrawn from the liquid and exposed to the air to favor the oxidation of the antimony, when, after a time, the deposit will be entirely dissolved as antimoniate of potash. On now removing the copper-foil, acidulating the liquid with hydrochloric acid, concentrating by evaporation, and treating with sulphuretted hydrogen, the *pentasulphide* of antimony will be precipitated of an orange-red color. The whole of the antimony may be thus removed, by using successive slips of copper.

Another mode of detecting antimony when in very minute quantity in the organs, is by a modification of the galvanic test (described on page 262), as recommended by Prof. Taylor (Prin. and Prac. of Med. Jurisp., 1873, p. 314). Coil a portion of pure zinc-foil around a portion of clean platinum-foil, and introduce the two metals into the hydrochloric acid decoction of the tissues, just sufficiently dilute to prevent too violent an action on the zinc. Warm the organic liquid, and suspend the coils in it. Sooner or later, according to the quantity of antimony present, the platinum will be coated with an adhering black powder of metallic antimony. Wash the platinum-foil, and digest it in strong nitric acid. So soon as the black deposit of antimony is dissolved from its surface, the platinum should be removed. Add a few drops of nitric acid, and evaporate to dryness. The residue redissolved in hydrochloric acid, and the solution diluted and treated with sulphuretted hydrogen, will yield the pure sulphide; or the black deposit on the platinum may be dissolved off by sulphide of ammonium (see p. 262).

The absorbed antimony may also be extracted from the tissues by means of hydrochloric acid and chlorate of potassa, as recommended for Arsenic (p. 246). In the case of antimony, however, there is no occasion to employ sulphurous acid or a sulphide. The precipitated sulphide should be purified as directed on page 269.

The *urine* should always be examined for absorbed antimony. The elimination of this metal by the kidneys commences very soon after it has been taken, and continues for some time after it has been discontinued (see p. 260). The urine should be evaporated nearly to dryness, when it may either be treated with hydrochloric acid and chlorate of potassa (see p. 246), and the antimony procured as a sulphide; or it may be boiled with water and hydrochloric acid, and tested by the copper, the tin, and the galvanic test, and by Marsh's process; or it may be carbonized by sulphuric acid and heat, and the antimony extracted as mentioned, under the head of ARSENIC.

CHLORIDE, OR BUTTER OF ANTIMONY.—This is a strong corrosive poison, and has caused death in several instances. Its symptoms and post-mortem appearances resemble those produced by strong hydrochloric acid. When thrown into water, it produces a copious, white, flaky precipitate, by which it is readily identified: when this is touched with sulphide of ammonium, it assumes an orange-red color. The clear liquid is proved to contain hydrochloric acid, by the addition of nitrate of silver.

CHAPTER XVI.

POISONING BY MERCURY.

IN the *metallic* state, mercury is not regarded as a poison. It has frequently been administered in large quantities by physicians in the treatment of constipation, in which affection it is supposed to act remedially by virtue of its gravity and liquid form—the metal passing rapidly through the bowels. The *vapor* of mercury is very poisonous in its effects. There is no doubt that this vapor passes off from the metal at ordinary temperatures, so that persons exposed to breathing it become speedily affected with mercurial salivation, or ptyalism. A noted instance of this fact is afforded in the case of the British ship *Triumph*, in the year 1809, which had received on board from a Spanish wreck a quantity of quicksilver contained in leather bags. In consequence of the defective storage of this cargo, some of the bags were broken, and the liquid mercury escaped into the hold of the ship. The effect was that in a very short time every living creature on board became salivated; even the lower animals, as cows, horses, and poultry, were thus affected, and many died.

Artisans who work in this metal, such as smelters of the ores, looking-glass platers, water-gilders, and barometer-makers, are very liable to become poisoned by the fumes. The symptoms of this sort of poisoning sometimes commence suddenly and at other times come on gradually; and they may or may not be accompanied by salivation. The general morbid condition thus induced is termed *mercurial tremors*, *shaking palsy*, and *tremblement mercuriel.* The upper extremities are commonly first affected, and then, by degrees, all the muscles of the body. There is a general unsteadiness of motion in the arms and legs, so that the patient cannot grasp any object or plant his foot firmly on the ground. In bad cases, he cannot either speak or masticate his food. If the disorder is not checked, it proceeds to a fatal termination, attended with a loss of memory, insomnia, and delirium. Another curious symptom, not generally recognized, although very constantly present, is a brittle state of the teeth, causing them to chip. (Guy's Forensic Medicine, 1868, p. 474.)

The proper preventive treatment of this affection consists in cleanliness and good ventilation, together with the free use internally of albumen in the form of white of eggs.

All the compounds of mercury are more or less poisonous; but the one of most medico-legal importance is corrosive sublimate.

CORROSIVE SUBLIMATE (*Protochloride of Mercury*).—This salt was formerly named *bichloride* of mercury. The difference in the nomenclature arises from the circumstance that the chemical equivalent of mercury was formerly taken as 200; whereas at present 100 is regarded as its true equivalent. According to this latter view, calomel must be considered as a *subchloride.*

Properties.—It occurs either in heavy, semi-transparent, crystalline masses, or as a white, amorphous powder. It has a peculiar, nauseous, styptic taste; it is soluble in twenty parts of cold and in two parts of boiling water. It is still more soluble in alcohol and ether: the latter solvent is employed to separate it from the aqueous solution.

Symptoms.—These generally come on immediately or very soon after the poison is taken. A nauseous, metallic taste is perceived in the act of swallowing. There is a sense of heat or constriction in the mouth and throat; nausea, and violent retching; vomiting of matters frequently tinged with bile and blood; pain in the abdomen, which usually is swollen, and tender to the touch; severe purging, sometimes of bloody matters, accompanied with tenesmus, as in dysentery; great anxiety; flushed and swollen countenance, though sometimes it is pale and anxious. The pulse is small, frequent, and irregular, and is scarcely perceptible when the symptoms become aggravated. The tongue is white and shriveled; the skin cold and clammy; the breathing difficult; intense thirst; scanty or suppressed urine; cramps of the extremities; stupor, fainting, convulsions, and death. The external parts of the mouth are found to be swollen, and often present a white appearance, as if the cavity had been washed with a solution of nitrate of silver; the lips are often swollen. In cases which do not prove rapidly fatal, salivation is usually superadded, as well as the painful train of nervous symptoms caused by the specific impression of mercury on the system.

There are occasional exceptions to some of the above-mentioned symptoms of poisoning by corrosive sublimate: thus, there may be an absence of abdominal pains, and also of vomiting and purging; and cases are reported in which the pulse underwent no change until just before death. In some instances the symptoms partially remit.

This poison differs from arsenic, according to Dr. Taylor (On Poisons, p. 399): 1, in having a well-marked taste; 2, in producing violent symptoms in a few minutes; 3, the evacuations are more frequently mingled with blood. If the patient survive several days, the symptoms resemble those of dysentery,—violent straining, and shreds of bloody mucus in the discharges from the bowels being frequently noticed.

The *external* application of corrosive sublimate has sometimes been followed by fatal consequences; and it is a remarkable fact that in such cases both the symptoms and the post-mortem lesions resemble very closely those attendant upon an ordinary case of poisoning by swallowing.

Dr. Vidal (Gazette des Hôpitaux, July, 1864) reports a case of a woman aged twenty-eight years, who accidentally had applied to the surface of her body the acid nitrate of mercury instead of a liniment. Besides intense inflammation of the skin, there were several large eschars upon different parts of the body; there was bilious vomiting, which was afterwards tinged with blood, abundant diarrhœa of a dysenteric character, cramps, extreme anxiety, great pain in the abdomen, suppression of urine, swollen and bloody gums, along with a bluish line at their junction with the teeth. Nervous symptoms appeared on the sixth day, together with extreme feebleness; death occurred on the ninth day. On examination, the interior of the stomach was found reddened, the vessels injected, and there were ecchymosed spots. A similar appearance was observed under the mucous coat of the bladder and throughout nearly the whole intestine. The blood was black and fluid. The microscopic examination of the kidneys disclosed considerable injection of the parenchyma, especially around the Malpighian bodies; the epithelial cells were deformed, granular, and partially destroyed. M. Flandin, by chemical analysis, obtained a sensible quantity of mercury from the liver, but not from the other organs.

Other cases of fatal result from the application of corrosive sublimate are on record. In one, the subject was a child, who died in about a week, after suffering the severest constitutional effects. In two others, also children, aged respectively seven and eleven years, an ointment composed of two drachms of corrosive sublimate to an ounce of tallow was rubbed upon the scalp for the treatment of *porrigo favosa.* Excessive suffering immediately ensued, and in forty minutes the children were completely delirious. They vomited continually a green-colored matter, and had great pain in the bowels, with diarrhœa and bloody stools. In the youngest, there was complete suppression of urine. Death occurred in one on the seventh, and in the other on the ninth day. There was no ptyalism. (Wharton and Stillé, Med. Jurisp., 1873, vol. ii. p. 407.)

A case of fatal poisoning by the external application of corrosive sublimate is reported in the London Chemist and Druggist, Sept. 15, 1871. For a child nine years of age suffering from ringworm of the scalp, a lotion containing two grains of the salt to a drachm of alcohol was prescribed, as an application to the shaven scalp, by means of a brush. This is stated by the physician to be the formula given by Dr. Tilbury Fox. The application at first gave no pain, but in the course of a few hours the child suffered greatly, the head and neck being also much swollen. The pain and swelling continued to increase in spite of every effort to check it, and the child died in the course of a few days. Only a single application was made. According to Dr. Fox, the lotion generally produces blistering of the skin, which is the design in using it, but it is not absorbed. In the present instance, however, absorption of the poison, unfortunately, took place, and with fatal effect.

Fatal dose.—The smallest dose reported to have destroyed life is three grains. Dr. Taylor considers that under favorable circumstances from three to five grains, or even less, would destroy an adult (On Poisons, p. 412). Very large doses have been taken with impunity, having been speedily vomited, or neutralized by antidotes promptly exhibited.

Fatal period.—The shortest fatal period on record is that of a case reported to Dr. Taylor, in which death occurred in *less than half an hour*, from an unknown amount of the poison. Some cases prove fatal within three or four hours; but generally life is protracted from one to five days.

In a summary of nine cases given by Prof. Guy (Forensic Med., 1868, p. 475), about half the number died in less than twelve hours, and the remaining half in a period varying from three to eleven days. In the case of this poison, as with arsenic, the fatal period is very variable.

Mortality.—More than half the cases.

Treatment.—If free vomiting has not taken place, the evacuation of the stomach should be aided by warm diluent drinks. The best antidote is *albumen*, as found in eggs. This decomposes the mercurial salt, forming an insoluble inert compound; but this is redissolved by a large excess of albumen. The white of one egg is generally supposed to be sufficient to neutralize four grains of corrosive sublimate. In the absence of eggs, gluten, or wheat flour made up into paste, may be employed. The free use of milk is also recommended.

The early and free use of albumen is nearly always attended with success. Various other antidotes have been recommended at different times, such as the *protosulphide of iron*, *iron-filings*, a mixture of *gold-dust* and *iron-filings*, as recommended by Dr. Buckler, of Baltimore, *protochloride of tin*, etc. None of these, however, can compare in value with albumen, which has stood the test of many years' experience.

Post-mortem appearances.—The mucous membrane of the mouth, throat, and gullet is often found softened, of a white or grayish color, and sometimes inflamed. The action of this poison upon the stomach and bowels is generally more decided than that of arsenic. The coats of the stomach may be found more or less corroded and softened, presenting a slate-gray color, which is by some ascribed to the deposit of fine reduced mercury. Sometimes dark gangrenous spots are observed. Similar appearances have been seen in the large and small intestines, especially in the cæcum. Sir R. Christison mentions a case where the patient survived thirty-one hours, in which there was perforation of the stomach. The urinary organs are generally found greatly inflamed, the bladder much contracted and empty, and the kidneys highly congested, especially about the Malpighian bodies, and the epithelial cells deformed, granular, and partially destroyed.

In *chronic or slow poisoning* with corrosive sublimate, the following symptoms are generally observed: a coppery taste in the mouth, loss of appetite, a fetid breath, tenderness of gums, pains in the stomach and bowels, nausea, inflammation and ulceration of the salivary glands, swelling of the tongue, increased flow of the saliva, a quick pulse, hot skin, great muscular debility, and emaciation. A bluish line is frequently observed around the edges of the gums, like that occasioned by lead-poisoning.

It may be well to remember that *salivation* is not an invariable attendant on acute mercurial poisoning; but it is nearly always observed in the chronic form. There is a very marked difference in the susceptibility of persons to the mercurial influence. In some, the smallest dose will speedily excite the most profuse ptyalism, as in a case mentioned by Sir R. Christison, where two grains of calomel caused ptyalism, extensive ulceration of the throat, exfoliation of the lower jaw, and death. It should not be forgotten that salivation may be caused by other substances besides mercury: among these may be mentioned iodide of potassium, digitalis, antimony, arsenic, etc.

In a suspicious case, the chemical detection of mercury in the saliva would settle the question as to its mercurial origin.

Chemical analysis.—I. *As a solid.*—Heated on platinum-foil, it fuses and is wholly dissipated in white, acrid fumes. If the vapor be received on a cool surface, it condenses in groups of peculiar-formed, white, radiating crystals. When touched with a drop of liquor potassæ, it turns of a brownish-yellow color: calomel, under similar treatment, becomes black. A solution of iodide of potassium immediately causes it to assume a bright scarlet color: this reaction is very delicate, and serves to detect the minutest fragment of the salt. If a drop of the iodide of potassium solution be placed upon a piece of bright metallic copper, in contact with the smallest portion of corrosive sublimate, the latter will be decomposed, and a bright silvery stain will be seen upon the copper, especially if it be rubbed with the finger or other soft substance; a drop of hydrochloric acid may be substituted for the iodide of potassium. This stain is immediately dissipated by heat. Sulphide of ammonium applied to a small portion of the powder at first turns it yellowish, but subsequently black. Heated in a reduction-tube along with dried carbonate of soda, a sublimate is deposited in the form of a white ring, which, under the microscope, is found to be composed of globules of metallic mercury. The white residue, when dissolved in water by the aid of heat and a little nitric acid, may be proved to contain *chlorine* by the action of nitrate of silver, which precipitates the white chloride of silver.

II. *As a liquid.*—(*a*) A drop of the solution, not very dilute, if evaporated on a glass slide, will yield a deposit of long, needle-shaped or prismatic crystals. The presence of organic matter readily interferes with the crystallization of this salt. (*b*) It is easily decomposed by albumen, fibrin, casein, gluten, and tannic acid: hence the usefulness of these articles as antidotes to this poison. (*c*) *Solution of potassa or soda* in excess throws down the yellow oxide of mercury. If not in excess, the precipitate has a brownish color. This precipitate may be identified by drying and heating in a reduction-tube: a sublimate of globules of mercury will be deposited, with the evolution of free oxygen gas. (*d*) *Ammonia* causes a white precipitate (ami-chloride of mercury): this is soluble in a large excess of the precipitant. If the precipitate be dried and heated, it volatilizes without residue, in which respect it differs from all other metallic precipitates produced by ammonia. (*e*) *Solution of iodide of potassium* causes a bright scarlet iodide of mercury, readily soluble in excess of the precipitant. At first the color is yellow, but it quickly becomes scarlet. When this iodide is dried, and heated in a reduction-tube, it volatilizes unchanged, and condenses in a yellow, semi-crystalline deposit, which slowly changes to a scarlet color. (*f*) *Protochloride of tin* in limited quantity causes with corrosive sublimate a white precipitate of the subchloride (calomel); if the reagent be in excess, a dark-gray precipitate of metallic mercury takes place, which runs into distinct globules on being boiled. (*g*) Sulphuretted hydrogen and sulphide of ammonium in minimum quantities produce, in a solution of corrosive sublimate, a precipitate, the color of which is gray or white, at first; on adding more of the reagent, the color changes to reddish and black, and finally becomes completely black (the black sulphide). This progressive change of color, under the circumstances mentioned, is peculiar to the persalts of mercury. Any of these precipitates, if dried and mixed with dry carbonate of soda (or with iron-filings, Orfila) and then heated in a reduction-tube, will yield a sublimate of metallic globules. (*h*) *The copper test.*—If a piece of bright copper-foil or copper wire be plunged in a solution of corrosive sublimate acidulated with hydrochloric acid, it speedily becomes coated with a bright silvery deposit of metallic mercury. When the copper is thoroughly dried, and heated in a reduction-tube, a sublimate of metallic globules will be obtained, easily recognized under the magnifier. This test is exceedingly delicate, and will serve to detect one ten-thousandth of a grain, if the deposit be received on a very small surface of copper and the latter heated in a very small reduction-tube. But even much more minute portions may be recognized, according to Wormley (Micro-Chem. of Poisons, p. 339), by the following method. A quite thin and perfectly clean tube of the diameter of about one-tenth of an inch is drawn out into a small capillary neck. The coated copper is then introduced through the wider portion of the cooled tube to the point of contraction, and the wider end very carefully fused shut by means of the blowpipe. The appearance of the tube then resembles that of a small thermometer-tube—the bulb containing the coated copper. The tube is now heated at the point containing the copper, and then the capillary end is closed. It is then wiped, and examined under the microscope, when a well-defined ring of mercurial globules will be seen in the narrow tube, a short distance above the contracted portion. The tube thus closed may be kept for future reference; though, after long periods, the sublimate gradually becomes fainter and finally disappears. This method has also the advantage of allowing the higher powers of the microscope to be applied, on account of the extreme thinness of their walls. According to Prof. Wormley, one five-hundred-thousandth of a grain, treated as above, yielded as many as twenty satisfactory globules of mercury, the largest of which measured one three-thousandth of an inch in diameter, though most of them averaged from one five-thousandth to one ten-thousandth of an inch. A power of about two hundred and fifty diameters is the highest that can be successfully used, on account of the curvature of the glass (*loc. cit.*, p. 340).

In case the metallic sublimate on the sides of the reduction-tube should be very faint and uncertain, M. Tardieu (Sur l'Empoisonnement, p. 580) recommends a neat and satisfactory method. A minute crystal of iodine is pushed into the tube, by means of a platinum wire, as far as the sublimate; the open end of the tube is then stopped up with wax, and it is kept in a horizontal position, at a temperature of 30° to 40° C. In about twelve hours, the deposit, if it be composed of mercury, will assume a lively scarlet tint, due to the production of the red iodide of mercury. After removing the fragment of iodine, the tube may be gently and progressively heated from below by the flame of a spirit-lamp, when the scarlet color will change to yellow, which latter hue will continue as long as the tube remains warm; but on cooling the tube, or on contact with a foreign body, it will resume its scarlet color.

(*i*) *The galvanic test.*—This consists in the employment of a strip of gold wound around a strip of zinc (tin or iron will answer in place of the latter), and introducing them into the mercurial solution, slightly acidulated with muriatic acid. In a short time the gold will be covered with a silver-colored film of metallic mercury. On washing the gold in water and ether, carefully drying it, and heating in a reduction-tube, the characteristic sublimate of mercurial globules may be obtained. A very simple method of employing the galvanic test, according to Prof. Guy (Forensic Medicine, p. 467), is to take a narrow strip of zinc, sufficiently small to be introduced into a reduction-tube, moisten it, and take up as much gold-leaf as will adhere to it; introduce this into the acidulated solution: the gold will soon be covered with a gray film. Wash and dry it carefully; introduce it into a reduction-tube and heat with the flame of a spirit-lamp; a ring of metallic globules will be formed. This is considered one of the most delicate of all the tests: it is generally employed in the detection of the poison in organic mixtures.

In the employment of the copper and the galvanic test, it must be remembered that it is not the silvery deposit upon the copper or gold that affords the proof of the presence of mercury, but only the actual obtaining of the *metal* in the form of globules, by sublimation.

In organic mixtures.—As corrosive sublimate is soluble, it is rare to meet with it in the solid form; but, since it may be administered in mass, some of the poison may be discovered among the other substances, by merely stirring the liquid, at the same time adding, if very viscid, distilled water; the corrosive sublimate, from its weight, will subside in lumps, and may be collected and identified. But, as this poison is readily decomposed by albumen, fibrin, gluten, tannic acid, and other substances, it will most probably be found in a state insoluble in water. Still, under these circumstances, sufficient of the mercury in the soluble condition can usually be detected in complex fluids, even after a considerable time. Hence we may expect to find this poison in both conditions —dissolved and in combination. In the former state, the liquid should be obtained clear by filtration, after boiling with water, if necessary. The solid matters should be pressed, dried, and set aside for future examination. The liquid portion should be slightly acidulated with hydrochloric acid, warmed, and tested with a slip of bright copper, the latter being allowed to remain in the liquid for some hours, if no deposit occurs in a short time. When the quantity of corrosive sublimate in the solution is large, it may be removed by means of ether. The filtered liquid containing the poison is put into a stoppered tube containing twice its volume of pure ether, and the contents thoroughly agitated at intervals. Allow the liquid to subside, remove the ether by means of a pipette, and allow it to evaporate spontaneously. As the ether passes off, the corrosive sublimate will be deposited in white, silky prisms. These may be purified, if necessary, by solution in water or alcohol, and again crystallized. Corrosive sublimate may thus be separated from arsenic and other mineral poisons.

Tardieu (*loc. cit.*, p. 581) objects to this mode of separation by ether, on two grounds: first, because the poison can never be wholly separated by this means; and secondly, because the ether must necessarily dissolve a considerable quantity of fatty matters, which are deposited by evaporation of the ether, along with the mercurial salt. It should, moreover, be remembered that any mercurial salt associated with an alkaline chloride, such as chloride of sodium, would be acted upon by ether in a similar manner: consequently, the single fact of the presence of corrosive sublimate in the ether is not positive proof that the poisoning was occasioned by this particular salt.

As regards the solid portions, supposed to contain the poison in combination with certain organic substances, they may first be boiled with distilled water for about half an hour, stirring frequently. When cold, filter, and concentrate by heat; then examine in the manner directed for the first liquid portion (p. 279). If no evidence of the presence of mercury is given, the solid matters should be boiled in water containing hydrochloric acid until complete disintegration occurs; filter when cooled, and test as above. Another method, sometimes pursued, is to dry the solid matters thoroughly and digest the dried residue in warm nitro-muriatic acid, by which the insoluble mass is converted into the soluble corrosive sublimate. The acid liquor must be evaporated to dryness, and the residue dissolved in distilled water, and filtered. The corrosive sublimate may now either be dissolved out by ether or at once tried with the protochloride of tin, or by the galvanic test.

In the tissues and urine.—The urine should be evaporated to dryness, and the dried residue treated by the process next to be described *for the tissues.*

From four to eight ounces of the liver, kidneys, spleen, or other organs to be examined, should be dried, broken up, and then boiled, until dissolved, in one part of pure hydrochloric acid and four parts of water. When cool, the liquid should be strained through linen, and the residue pressed. The liquid, if in large quantity, should be concentrated by gentle evaporation, and, while still warm, a small piece of pure copper-foil should be introduced. The copper will acquire a coating of a silvery or silver-gray appearance in the course of a few minutes, or after a longer time. It may be removed, washed in water and alcohol, and examined by a magnifier, which will serve to distinguish the deposition of any metallic film. The copper is next to be rolled up and heated in a reduction-tube, when the deposition of metallic globules in the form of a ring, on the inside of the tube, will establish the presence of mercury in the original acid solution.

If arsenic had been present in the tissues at the same time with the mercury, the boiling of the acid liquid upon the copper would cause a deposit of both metals upon the latter; and when this is heated in the reduction-tube there will be seen a mixture of mercurial globules with octahedral crystals of arsenious acid in the sublimate. In the *cold* acid liquid arsenic would not be deposited on the copper, while mercury would be, at all temperatures. By this means we may separate these two metals when they exist in the same solution.

Dr. Taylor (On Poisons, p. 420) mentions a case of this nature where arsenic had been criminally administered to the deceased, and where also two grains of calomel had been taken, medicinally, two days before death. Twenty-one months after burial, the presence of both arsenic and mercury was distinctly proven through the sublimates obtained by means of the copper process.

It must not be forgotten that a person may die from poisoning by corrosive sublimate and yet no mercury be found in the tissues. Dr. Taylor cites two cases of this character: one of these patients survived fifteen days, after taking a large dose of the poison in whisky. Although the local effect on the mouth, throat, stomach, and bowels was of the most intense kind, the chemical analysis could detect no trace of mercury in any of the viscera: it had been entirely eliminated (Med. Gaz., vol. xlvi. p. 253). It also occasionally happens that this poison, like many others, cannot be detected after death, in the stomach. Taylor mentions several instances of this character (*loc. cit.*). In two, death occurred on the *fourth* day, after taking two drachms of the poison; in another, two drachms proved fatal in *six* days; and in a fourth, three drachms destroyed life in *six* days. In none of these could the poison be discovered in the stomach or intestines.

Many other metallic poisons are doubtless eliminated from the body through the saliva; a chemical examination of this secretion might often lead to their ready detection.

The above-described examination of the tissues and secretions can merely establish the presence of mercury, but not in the particular form of corrosive sublimate: the proof of the latter must be sought in the stomach and intestines, either by *dialysis*, or by the action of ether on the aqueous solution obtained from these last-mentioned organs. It must not be forgotten that the mere detection of small quantities of mercury in the tissues, organs, and secretions is no evidence of poisoning by this substance, unless it be supported by the character of the symptoms before death, and by the post-mortem lesions. The administration, before death, of a dose of calomel, blue-pill, etc., could readily account for the presence of mercury in the different viscera after death, on the well-recognized principle of absorption.

Allusion has been made to the importance of analyzing the *saliva* in a case of suspected mercurial ptyalism. This is readily effected by acidulating that secretion with one-fourth part of muriatic acid; a piece of bright copper attached to a platinum wire should then be immersed in it, and the whole kept at a warm temperature for several hours. If mercury be present in the saliva, the copper will become whitened. By heating the copper, after washing and drying, in a reduction-tube, the sublimate of characteristic mercurial globules will establish the proof of the presence of this metal.

The exact relationship between *salivation* and poisoning by corrosive sublimate is a matter of considerable medico-legal importance. It is well known, in the first place, that there is no fixed or definite period at which ptyalism comes on, after the poison has been swallowed. In some cases of acute poisoning it does not appear at all; and very rarely, unless the patient survive two or three days. Thus, in a case which occurred to Dr. Venables, in which two drachms of this substance had been taken, and the woman survived eight days, there was no salivation. But in another case, reported by Mr. Wood (Edin. Med. and Surg. Jour., li. 141), in which half a teaspoonful of the poison had been swallowed, salivation was profuse in the course of a few hours. Dr. Percy relates a case in which the saliva was flowing profusely one hour and a half after a woman had taken a dose of thirty grains (Med. Gaz., 1843, i. p. 942). In these cases of early salivation, it is alleged that the fetor of the breath is absent, and that the abundant flow of saliva is to be ascribed to the local irritant effect of the poison, rather than to the result of absorption. It must certainly be admitted that the direct irritant action of corrosive sublimate upon the tongue and mouth may cause a profuse discharge of saliva, with swelling of the lips. (See case reported in Prov. Med. Jour., Nov. 18, 1843, p. 127.)

Another point for consideration is the difference in the susceptibility of persons to the mercurial impression. As a general rule, children are less susceptible than adults; the robust, less than the delicate. Certain morbid conditions of the system particularly predispose to its injurious impression, among which may be mentioned albuminuria and anæmia. In an apoplectic patient of Dr. Bright's, five grains of calomel placed on the tongue produced, in three hours, violent salivation, and such swelling of the tongue as to render scarification necessary. Three grains of corrosive sublimate in three doses have caused violent ptyalism; three five-grain doses of blue-pill, one every night, have proved fatal; two grains of calomel have caused ulceration of the throat, exfoliation of the jaw, and death; and the external application of three drachms of mercurial ointment has destroyed life in eight days. (Guy's Forensic Medicine, 1868, p. 471; from Christison on Poisons.)

It is generally admitted that mercurial salivation may be intermittent; that is, it may cease for a time, and reappear without any mercurial preparation being given in the interim. Such cases are, however, rare; but the fact of the possibility of their occurrence is of importance in connection with medical jurisprudence. One case of this kind is quoted by Mr. Swan (On the Action of Mercury, 1847, p. 4), in which salivation recurred after an interval of six months.

Another fact of importance to the legal physician is, that salivation may be produced by other agents besides the preparations of mercury; so that this symptom when taken alone can never furnish evidence of poisoning by mercury. Salivation may arise *spontaneously*, from disease of the salivary organs, or even from a mechanical cause, such as the irritation of a set of artificial teeth. It not unfrequently follows the internal use of iodide of potassium, iodine, the preparations of copper, bismuth, lead, antimony, gold, arsenic, digitalis, croton oil, cantharides, colchicum, and other articles of the materia medica. The usual diagnostic points of difference between mercurial and non-mercurial salivation are said to be the presence of the coppery taste, the fetor of the breath, and the spongy and ulcerated condition of the gums in the former, while they are wanting in the cases of salivation occasioned by other causes. But it would seem that these characters have been equally met with in the salivation produced by arsenic and by bismuth. (Prov. Med. Jour., Oct. 22, 1845, p. 638; also, Lancet, June 13, 1846, p. 654.)

The disease named *cancrum oris*, or *canker of the mouth*, not unfrequently occurs among children brought up under bad hygienic influences, or while recovering from exhausting diseases, especially from hooping-cough and measles. Among the symptoms of this affection are ulceration of the gums and a falling out of the teeth, along with gangrene of the cheek. In such cases, if a preparation of mercury should have happened to be given, it might easily become a serious question to determine whether death actually resulted from mercury acting as a poison, or from the disease. Dr. Taylor (On Poisons, p. 406) cites a case in point. A charge was made against a medical practitioner of having caused the death of a child, aged four years, by administering an overdose of some mercurial preparation, for the treatment of hooping-cough. On the fourth day, the child complained of soreness of the mouth, the teeth became loose and fell out, the tongue and cheek were much swollen; and the child died in the course of a few days, from gangrene of the left cheek. The answer to the charge was, that not a particle of mercury had been exhibited,—a fact clearly proved from the prescription-book of the medical attendant! This, then, was an instance in which gangrene from spontaneous causes had been mistaken for mercurial poisoning. Had the medicine prescribed contained any mercury, a verdict affecting the character of the practitioner would probably have been returned. An important case of this character, in which the medical witness relied upon the "mercurial fetor" as characteristic and distinctive, will be found in the "Lancet," June 13, 1846, p. 654 (*loc. cit.*). As before observed (p. 285), the chemical analysis of the *saliva* would settle any question of this kind.

Quantitative analysis. — Corrosive sublimate may be estimated as a *sulphide*, by first carefully washing and drying the precipitate obtained by sulphuretted hydrogen. Every 100 grains of the dried sulphide are equivalent to 116.81 grains of anhydrous corrosive sublimate. By employing the chloride of tin with a known quantity of the mercurial solution, the metal is precipitated in the form of globules: it should be purified, by first boiling it in a solution of potassa, and afterwards in hydrochloric acid. Every 100 grains of metallic mercury are equivalent to 135.5 grains of corrosive sublimate.

The other compounds of mercury which may occasionally prove poisonous are *red precipitate*, *white precipitate*, *red oxide*, *cinnabar* or *red sulphuret*, *calomel*, the *nitrates*, and the *sulphates*.

A case of poisoning by red precipitate is reported in the "Irish Hospital Gazette," Oct. 15, 1873, that of a girl, aged fifteen years, who swallowed half an ounce by mistake. No urgent symptoms were manifested; an emetic of sulphate of zinc was administered, and the liberal use of milk enjoined as a diet. The following day, her lips, gums, and mouth were very sore, swollen, and reddened; there was the mercurial fetor from the breath, with headache, and pains in the epigastrium. She took Battley's sedative mixture, together with an alum gargle for her mouth; poultices were applied to the abdomen. Improvement commenced immediately; and, after a dose of castor oil, she had no further trouble, except a soreness of the mouth for about a week, and the loss of two front teeth.

CHAPTER XVII.

POISONING BY COPPER.

Copper in the metallic state is not poisonous. But if taken into the stomach, it is soon acted upon by the secretions and contents of this organ, and soluble compounds are generated which may prove highly poisonous. Copper coins are sometimes swallowed by children, and may produce injurious effects both by their mechanical irritation, and by the poisonous action of the resulting compounds. Many instances of this character have been recorded, in some of which colicky symptoms, irritation of the bowels, nausea and vomiting, emaciation, and death have resulted. Such cases sometimes are protracted for several years.

Instances of accidental poisoning by inhaling copper alloy in fine powder are afforded in persons who are engaged in what is termed printing in gold. The alloy is applied on the sized letters in the state of an impalpable powder, so light that it floats in the atmosphere, and is constantly being inhaled into the lungs of the workmen. The symptoms produced are vomiting of a greenish fluid, heat and constriction of the gullet, pain in the stomach, loss of appetite and rest, and a severe itching in all the parts covered with hair; these, on examination, are found to be of a deep-green color. (Falconer on Copper-Poisoning.)

Cases of accidental poisoning by copper are very frequently to be traced to want of cleanliness in cooking, or to keeping food in copper vessels. Many kinds of food are contaminated by contact with copper, especially such as contain a vegetable acid, as vinegar, an alkaline chloride, as common salt, or any kind of oil or fat. Hence pickles, or preserves in acid, are soon impregnated with copper under such circumstances; so, also, salt provisions, as fish, etc., very soon acquire poisonous properties if kept in copper vessels; likewise, articles of food of a greasy or fatty nature soon become affected with the poisonous contamination. In all these cases the poisonous impression appears to be produced chiefly about the line of contact between the particular substance and the external air, as is denoted by a greenish discoloration. So long as the copper utensils employed in cooking are kept perfectly clean and bright, no risk is incurred in their use; but if cleanliness be neglected, and a deposit of the green carbonate be allowed to accumulate, this latter, being a very poisonous salt, will impart very noxious properties to the food which may happen to come in contact with it.

Dr. Falconer found that distilled water kept several weeks on a polished plate of copper neither injured its lustre, nor acquired any taste, nor became colored by ammonia; and Drouard afterwards observed that distilled water, kept for a month in copper filings, did not contain any of the metal. If, however, the water contain common salt in considerable proportion, it will certainly become contaminated (Christison on Poisons, p. 450). Eller has shown that milk, tea, coffee, beer, and rain-water, kept in a state of ebullition for two hours, do not contract the slightest impurity from clean copper; the same is true (according to Falconer) of turnips, cabbage, potatoes, carrots, onions, rice, and barley (*ibid.*).

Whilst no danger might result from boiling acidulous liquids in *clean* copper vessels, it would be very unsafe to keep these fluids *cold*, in the same vessels. In the latter case the atmospheric air begins to act, while in the former it was expelled by ebullition. It has been observed that in preparing food or preserves in copper vessels, it is only when the fluid ceases to cover the metal, and is reduced in temperature, that solution of the metal begins. Inattention to this difference has been the cause of fatal accidents, of which the following case will serve as a good example. A servant left some sour-krout for only a couple of hours in a copper pan which had lost its tinning. Her mistress and daughter, who ate of the cabbage, died after twelve hours' illness. Wildberg found the cabbage so strongly impregnated with copper that it was detected by metallic iron. (*Ibid.*, p. 452.)

It may be inferred from the preceding observations that there is hardly any article of food or drink which may not be contaminated, if kept in copper vessels, as there are few articles that do not contain either an acid or some fatty matter; and it further appears that the impregnation scarcely ever takes place during the boiling of these articles, but only during the preservation of them in the cold state. For ordinary use, it would certainly be safer to dispense altogether with copper vessels, and substitute iron or tin. Indeed, copper vessels are now usually tinned, to prevent accidental impregnation. As the tinning consists of an alloy of tin and lead, it might happen, according to Dr. Taylor, that lead-poisoning may thus be substituted for copper-poisoning. As most of the copper of commerce contains arsenic, it might readily happen, in the use of copper vessels for culinary purposes, in which the metal is converted into cupreous compounds by means of acids and fats, that arsenic might be found in the green incrustations. Dr. Taylor states that he has never found any *dissolved* arsenic, where the copper thus forms an insoluble salt. This author also alludes to an impure gold alloy, largely composed of copper, being used by the lower class of dentists for forming the plates for the support of artificial teeth. These have been known to affect seriously the health of persons wearing them; the secretions of the mouth favoring the production of poisonous salts of copper, and probably, also, liberating arsenic. (Med. Jurisp., Am. ed., 1873, p. 176.)

Another source of accidental poisoning by copper is mentioned by the English and French writers, in the use of an alloy resembling gold for ornamenting cakes and confectionery; and also of blue and green papers colored by copper for wrappers of bonbons, etc. The alloy may readily be distinguished from gold by its solubility in nitric acid, which gives a blue-colored solution. In France, an ordinance prohibits the use of the colored wrappings above mentioned, on account of their poisonous nature. Still another source of contamination by copper has been pointed out as occurring on the continent of Europe, and especially in France, namely, the adulteration of bread with a very small quantity of sulphate of copper, with the twofold purpose of making the bread whiter and at the same time increasing its weight, by causing the dough to absorb more water. Orfila proved this to be the case by incinerating several loaves of bread, and detecting copper in the ashes. Still, as Tardieu recommends, in any case of suspected adulteration of bread with copper, where the latter exists only in minute quantities, it will be well first to ascertain whether it might not have been accidentally introduced by means of the flour or water employed. In one instance it was traced to the meal, which had been contaminated from the copper cylinders used to grind it.

In the making of many preserved fruits and pickles, the salts of copper (blue vitriol and verdigris) are quite frequently employed to impart a fine green color to them. This, of course, is a very pernicious practice, and ought to be discountenanced. Dr. Hassall states that he found copper in sixteen different samples of London pickles, and it was most abundant in those that were green (Food and its Adulterations, p. 388). Dr. Taylor detected copper in a sample of preserved green gooseberries that had produced symptoms of poisoning in a child.

An easy method of detecting the adulteration is to place the pickles or fruit in a solution of ammonia, when, if copper be present, the substance will turn them blue. A simpler method is to plunge a bright needle into the pickle or other substance: if copper be present, it will speedily receive a reddish coating; this may easily be examined by ammonia.

All the salts of copper are poisonous; but those of medico-legal importance are the *sulphate* (blue vitriol or blue-stone) and the *subacetate* (verdigris). The *arsenite* (Scheele's green) and the *aceto-arsenite* (Schweinfurt green) have already been described under ARSENIC. What is sometimes termed *natural verdigris* is the carbonate of copper, which is produced by the action of moist air on the metal: this is first slowly oxidized, and then converted into a carbonate by the carbonic acid of the atmosphere. It constitutes the greenish incrustation that collects upon copper or brass vessels which are not kept properly cleaned.

It is seldom that the copper salts are administered as poison with a homicidal intention, since they are so easily detected by their color and taste. Neither would they be likely to be accidentally taken, for the same reason: occasionally they have been employed suicidally.

Symptoms of acute poisoning.—Blue vitriol may be taken as the type of the copper salts. This has frequently been given as an abortive. In a large dose it speedily produces very violent vomiting, which may have the effect of expelling the whole of the poison from the stomach, whereupon the patient recovers. There is headache, pain in the abdomen, of a colicky character, with purging. The vomited matters are distinguished by being of a blue or green color; the latter tint might be owing to the presence of bile, in which case it will not acquire a blue color on the addition of ammonia, which it would do if the green color were due to copper. In bad cases there are severe cramps, and sometimes convulsions. Dr. Percival met with an instance in which violent convulsions were produced in a young woman by two drachms of the sulphate of copper. The pulse is small, frequent, and irregular, and there may be great dizziness, with difficulty of breathing, anxiety, cold sweats, *extreme thirst*, and suppression of urine. Paralysis, insensibility, and even tetanus have preceded death, when the poison was administered to animals. Among the occasional symptoms in man may be mentioned jaundice, which, according to Sir R. Christison, is never observed in poisoning by either arsenic or corrosive sublimate.

The symptoms of *slow* poisoning by copper are generally caused by its accidental introduction into the system in articles of food, as already mentioned. These are much the same as those just described, although varying in degree. According to Orfila, there is an acrid, styptic, coppery taste in the mouth; parched and dry tongue; a sense of strangulation in the throat; coppery eructations; continual spitting; nausea and vomiting or retching; shooting pains in the stomach, often severe; gripings; diarrhœa of bloody or blackish stools, with tenesmus; debility, with anxiety in the præcordia; small, tense, irregular, and frequent pulse; heat of skin; great thirst; cold sweats; scanty urine; headache, vertigo, faintness, cramps of the legs, and convulsions.

(Toxicologie.) Another occasional symptom is a green line on the margin of the gums.

Fatal dose.—This has not been positively established. In Dr. Percival's case of the young woman mentioned above, *two drachms* of the sulphate occasioned most violent convulsions, although the patient recovered. Dr. Taylor mentions an instance where *half an ounce* of verdigris destroyed the life of a woman in sixty hours; and another of a child, where about twenty grains of the subchloride caused death (On Poisons, p. 524). Dr. Beck quotes a case of a man, aged forty years, who destroyed his own life by taking *one ounce* of sulphate of copper: it proved fatal in twelve hours (Med. Jurisp., ii. p. 667). In another instance, seven drachms of blue vitriol with three drachms of sulphate of iron caused the death of an adult in three days. On the other hand, cases are reported where an ounce and upwards has been swallowed, without a fatal result. The usual *medicinal* dose of the sulphate as an emetic is five to fifteen grains; and this may be repeated until emesis is effected.

Fatal period.—This is subject to considerable fluctuation. In the instance mentioned by Sir R. Christison of a mother and daughter being poisoned by sour-krout that had been exposed to contact with copper, the latter died in *twelve hours*, the former an hour later. A child, aged sixteen months, died from the effects of an unknown dose of solid sulphate of copper in *four hours:* this is the most rapidly fatal case on record. Usually, death does not occur for several days.

Treatment.—Free vomiting should be promoted by the copious use of warm mucilaginous drinks; the stomach-pump may be employed, if necessary. The best *antidote* is albumen, in the form of white of eggs, very freely taken. It acts, as in the case of corrosive sublimate, by forming an insoluble *albuminate.* Milk is asserted to be equally effectual with albumen: it acts by forming an insoluble *caseate* of copper. All the other reputed antidotes, such as iron-filings, ferrocyanide of potassium, magnesia, etc., are of much inferior value.

Morbid appearances.—These indicate a powerful irritant action on the alimentary canal, which is almost the exclusive seat of pathological change. In acute cases, the inside of the stomach and bowels often exhibits a bluish or greenish appearance, due to the color of the salt taken: it should not be forgotten that a somewhat similar appearance may be caused by altered bile. The proper method of distinguishing between them has been pointed out above (p. 293). The lining membrane of the stomach is generally inflamed and softened, and has occasionally presented an ulcerated, and even gangrenous appearance. The gullet has also shown signs of inflammation. The intestinal lining membrane exhibits a similar appearance; and cases are reported where the intestines were perforated, with the escape of their contents into the cavity of the abdomen. The intestinal tube is usually much distended with gas. According to Tardieu (Sur l'Empoisonnement, p. 525), there is a remarkable absence of the bloody extravasations, or ecchymoses, in the alimentary canal and in other places, which are so characteristic of some other irritant poisons. The lining membrane of the alimentary canal has been found throughout of a deep-green color, owing to small particles of verdigris adhering to it. In some cases of fatal poisoning, the stomach and intestines have been found in a perfectly natural condition.

Chemical analysis.—Metallic copper is easily recognized by its *red* color, and by nitric acid, which acts upon it with great energy, and converts it into the blue nitrate of copper. All the salts of copper are either of a blue or a green color, by which they may generally be identified. Only a few other metallic salts are thus colored: thus, some of the salts of cobalt are blue, and some of those of nickel, chromium, and uranium are green. Any of the salts of copper, when heated in the inner (reducing) flame of the blowpipe, impart a beautiful green color to the flame, and when mixed with dry carbonate of soda and heated on charcoal, the same flame reduces them to metallic copper, in globules, which may readily be identified.

The *sulphate*, or *blue vitriol*, occurs in large, beautiful, blue crystals, effervescent, very soluble in water, having a nauseous, metallic, styptic taste. Its solution has a blue color, recognizable when diluted to one five-thousandth of its weight. The solution has an acid reaction.

The *verdigris* of commerce is usually a mixture of the subacetate with other acetates of copper. It generally occurs in masses of a pale-green or bluish color. It has an unpleasant, acetous odor, and a nauseous, styptic taste. It is not entirely soluble in water, but deposits an insoluble basic acetate. It is completely soluble in dilute nitric and hydrochloric acids. Sulphuric acid decomposes it, forming the sulphate of copper, and liberating acetic acid, which is recognized by its characteristic odor.

The proper tests for copper *in solution* are the following: (1) *Ammonia* produces in solutions of a copper salt a bluish-white, amorphous precipitate—hydrated oxide of copper, which is immediately dissolved by an excess of the precipitant to a clear, dark, purple-blue solution. With very dilute solutions, ammonia may fail to yield a precipitate, but it immediately causes the liquid to assume a blue color: this color is at once destroyed on adding an acid. This test will readily distinguish one ten-thousandth of a grain of the sulphate in a drop of water (Wormley). This reaction is quite conclusive, provided the absence of salts of nickel, cobalt, and chromium be secured: thus, the salts of nickel yield with ammonia a partial precipitate of a light-green, hydrated oxide of nickel, readily soluble in excess of the reagent, forming a deep-blue solution. Cobalt gives with ammonia a blue precipitate, soluble in excess, and forming a reddish-brown liquid. Sesquioxide of chromium yields a bluish-green precipitate, soluble in excess, and forming a pink solution.

(2) *Ferrocyanide of potassium* throws down in moderately strong solutions of a salt of copper a copious reddish-brown precipitate of ferrocyanide of copper, insoluble in an excess of the reagent, and also in acetic and hydrochloric acids, but sparingly soluble in ammonia to a bluish-green liquid, from which it is reprecipitated by acetic acid. If the copper solution be very dilute, no precipitate may fall, although the liquid will assume a decided reddish-brown color. According to our experience, this test is even more delicate than the

ammonia test. Dr. Wormley says that even less than a one-hundred-thousandth solution can be detected by it. This reagent will act upon the violet-blue solution produced by ammonia, provided it be diluted, and a few drops of diluted sulphuric acid be added to neutralize the ammonia: one portion of the liquid may thus be tried by the two tests.

The salts of *uranium* also give a reddish-brown precipitate with ferrocyanide of potassium, but the uranium precipitate is changed to a *yellow* compound on adding an excess of ammonia, while the copper precipitate is changed to a *bluish-green* liquid. Besides, the uranium salt gives with ammonia a *yellow* precipitate, *insoluble* in an excess of the reagent, which is totally different from the behavior of a copper salt with ammonia. As the salts of copper are very apt to contain iron, the presence of the latter may modify the action of ferrocyanide of potassium, by imparting a *blue* tint to the precipitate, so as to disguise it.

(3) *Sulphuretted hydrogen* and *sulphide of ammonium* produce in solutions of copper a deep brownish-black precipitate, the sulphide of copper, even in acid solutions. If the solution is weak, the precipitate has a light-brown color. This precipitate is slightly soluble in an excess of sulphide of ammonium, but insoluble in fixed alkaline sulphides, and in the caustic alkalies. It is only sparingly soluble in *cold* concentrated nitric acid, but is readily dissolved in the *hot* acid, even when somewhat diluted, forming the blue nitrate, together with a little sulphate of copper.

The delicacy of this test is about equal to that of the two former ones. It should be remembered, however, that other metals besides copper are precipitated of a *brownish* color by sulphuretted hydrogen: consequently, the suspected sulphide requires the corroborative proof by hot nitric acid: the resulting blue solution is to be evaporated to dryness, dissolved in pure water, and then subjected to the foregoing tests.

(4) *The iron test.*—This is a simple and very satisfactory test, inasmuch as it produces the poison sought, *in the metallic condition.* It consists in immersing a piece of bright iron or steel in a slightly acidulated solution of a copper salt. Sooner or later, according to the strength of the solu-

tion, the latter will be decomposed, metallic copper being precipitated upon the iron, while a portion of the latter replaces the copper in the salt, becoming combined with its acid: thus, if it was a solution of the sulphate of copper, there would result a *sulphate of iron*, after the precipitation of the copper. The thickness of the deposit of the copper, and consequently the delicacy of the test, will depend very much upon the extent of surface over which the metal is distributed. Hence, in very dilute solutions, only a very small surface of iron should be exposed: a small, bright sewing-needle answers well on such occasions. If the needle be left some days in the solution, the iron will be slowly removed, and a hollow cylinder of metallic copper will remain. This may be dissolved in dilute nitric acid, and tested with the foregoing tests. Or the coated needle may at once be immersed in a small quantity of ammonia, and exposed to the air; the liquid then slowly becomes blue. According to Dr. Taylor, half a grain of sulphate of copper dissolved in a pint of water may thus be easily detected. Orfila, long ago, proposed to substitute phosphorus for polished iron: this substance effectually separates copper from its solutions, even when in contact with organic substances.

(5) *The galvanic test.*—This test, like the preceding, gives us the copper in the *metallic* form. If a few drops of a copper solution, slightly acidulated with sulphuric or hydrochloric acid, be placed in a platinum dish, or on platinum-foil, and a piece of bright zinc be then introduced, so as to touch the platinum through the liquid, metallic copper in its well-known red color is immediately deposited on the platinum. When the quantity of copper is small, there is merely a brown stain; but on pouring a few drops of ammonia upon it, and exposing it to the air, a blue liquid is slowly formed. A coil of fine platinum and zinc may be substituted for the other arrangement. The copper deposit may also be proven by dissolving it off the platinum by means of dilute nitric acid, evaporating to dryness, moistening with water, and testing as before described.

(6) *The blowpipe*, as already mentioned, may be used with great certainty for identifying copper in the *metallic*

state, in any suspected solid. A fragment of the substance, previously mixed with an excess of dry carbonate of soda, is placed upon a charcoal holder, and the inner flame of a mouth-blowpipe is made to play upon it. The metal is reduced; and on removing the cooled saline mass from the charcoal, pulverizing in an agate mortar, and washing off the powdered charcoal, minute beads or globules of the metal are procured, having the red color and other characteristic marks of this metal.

The above tests are amply sufficient to establish the presence of copper under all circumstances. Some others, however, are mentioned by authors, such as *potassa*, *arsenite of potassa*, *ferricyanide of potassium*, *iodide of potassium*, *chromate of potash*, etc., which are of much less value than those before described.

In organic mixtures.—The oxide of copper is liable to be precipitated from its salts by certain organic principles, such as albumen, casein, fibrin, and mucous membrane; but, as the resulting compounds are easily redissolved by acids and other matters, we may expect to find a portion, at least, of the copper dissolved. In such cases the liquid is usually of a bluish or greenish color, and has a strong coppery or metallic taste, even when the amount of the copper salt is far below the poisonous proportion. A portion of the clear liquid, after concentration, if deemed best, may be tested by immersing into it, previously acidulated, a bright sewing-needle, for several hours. Any reddish deposit upon it should then be tested as directed for the *iron test* (p. 297). It should be remembered, in the application of this test, that the needle may acquire a reddish coating, independently of the presence of copper, simply of the *oxide of iron;* but this may usually be distinguished by means of a hand-lens, though with greater certainty by means of ammonia, or nitric acid (see p. 296). Another caution to be observed in applying the iron test is, to avoid acidulating the liquid too strongly before immersing the iron; otherwise the deposit will be black, and the result unsatisfactory. If the foregoing trial test reveals the presence of a large amount of copper, the remainder of the liquid may be subjected to the action of sulphuretted hydrogen, and the precipitated sulphide of copper be decomposed by hot nitric acid, and the solution of the nitrate tested in the manner before described.

If, however, the copper is present in very small quantity, probably the best method of proceeding is to employ the *galvanic* test, as follows. A portion of the filtered liquid, acidulated with sulphuric acid, is placed in a platinum capsule or crucible, and a strip of zinc-foil is introduced so as to touch the platinum successively over its whole surface. At the different points of contact, metallic copper is deposited, until all the inside is coated over with the metal. The liquid is now removed, and the capsule well washed out. The deposited copper is next dissolved out by dilute nitric acid, and the resulting solution tested as already mentioned. Should the trial test just spoken of fail to detect the presence of copper in the liquid portion of the material submitted to examination, there can be little doubt of its entire absence. Still, the solids separated from the liquid by filtration may be boiled, with water and a little hydrochloric acid, for about fifteen minutes, and the solution obtained examined either by the iron test or by sulphuretted hydrogen.

It has already been stated that the sulphate of copper, as found in commerce, sometimes contains arsenic. Dr. Taylor states that ten grains of the crystals will be sufficient to yield evidences of this adulteration. Even when the sulphate has been given as an emetic, traces of arsenic have been discovered in the matters vomited, or in the contents of the stomach. (Med. Jurisp., Am. ed., 1873, p. 175.)

The stomach and its contents.—The inside of the stomach and intestines should always be carefully examined for small particles of the sulphate or acetate of copper, which are indicated by bluish or greenish discolorations. The contents of the stomach having been carefully collected in a clean porcelain dish, the inside may be thoroughly scraped, and the scrapings mixed with the contents; or the organ may be cut up into small pieces, and the whole, with the addition of pure water, if necessary, strongly acidulated with hydrochloric acid, boiled at a gentle heat until the solid matters are broken up. When cooled, the mass is filtered, the filtrate concentrated, and again filtered. A polished sewing-needle may now be used as a trial test; or the galvanic test (p. 298) may be so employed. A stream of sulphuretted hydrogen should now be slowly passed through the liquid, and the deposit, after standing for some hours, should be collected on a filter, well washed and dried, and then dissolved, by the aid of heat, with dilute nitric acid. The solution may be evaporated to dryness, the residue heated with a little distilled water and subjected to the appropriate tests; or a few drops of concentrated sulphuric acid may be added to the nitric solution, which is then to be evaporated to dryness, when the copper, if present, will remain in the form of the sulphate. On dissolving this in distilled water, and filtering, the usual tests may be applied.

If the precipitate by sulphuretted hydrogen contains much organic matter, the latter must be got rid of before the final testing. To this end, the residue obtained after solution in nitric acid and evaporation to dryness is moistened with concentrated nitric acid, and heated until the organic matter is entirely destroyed. If necessary, the process should be repeated. The dry mass is now treated with a little dilute nitric acid, again evaporated to dryness, the residue dissolved in pure water, and the tests applied as before.

Detection in the tissues.—Copper has been detected in the different organs and tissues of the body, after administration, by various observers. It remains in the organs much longer than arsenic and some of the other poisons,—as long as sixty days in the liver and lungs, according to M. L. Orfila. It is eliminated by the urine from the commencement of the active symptoms, but disappears from this secretion very soon after it has ceased to be taken; in which respect also it offers a contrast with arsenic.

In the examination of the organs for absorbed copper, different methods are recommended. The object in all of them is to effect the complete destruction of the organic matter; and, as copper is not a volatile metal, this can readily be accomplished without danger of loss. A portion of the liver, for example, should be cut up into small pieces, thoroughly dried, and incinerated in a porcelain capsule; the residuary ash should be digested in pure hydrochloric acid by heat, and then evaporated nearly to dryness. The residue may be dissolved in a small quantity of pure water, and examined by the usual tests, particularly by the polished needle.

Or, the solid matter, in pieces, may be mixed with nitric acid diluted with several volumes of water, and the mixture gently heated, with the occasional addition of chlorate of potassa, until the destruction of the organic matter has been accomplished, as indicated by the liquid becoming perfectly clear. It should then be diluted with water, and, after cooling, be filtered, and evaporated to dryness. The residue contains the copper, if present, as a nitrate, mixed with some organic matter. This is again put into a porcelain capsule, covered with pure nitric acid, along with chlorate of potassa, and moderately heated until it is perfectly dry; the heat is then urged to redness until the organic matter is completely destroyed, when the mass becomes almost white. On boiling this residue in nitric acid, slightly diluted, any copper present, together with the iron, which is usually found in minute quantities, will be taken up as nitrate. The solution should now be carefully evaporated, to expel the excess of nitric acid, and the residue dissolved in a little warm water, and tested. If it is desired to get rid of the iron that may be present, this metal may be separated by adding to the final solution ammonia in excess, which will precipitate the oxide of iron and leave the copper in the deep-blue solution. On filtering, and adding acetic acid to the filtrate, and afterwards ferrocyanide of potassium, the characteristic reddish-brown ferrocyanide of copper will be precipitated. Or, the iron may be separated by acidifying the final solution with hydrochloric acid, and transmitting sulphuretted hydrogen through it: all the copper will be precipitated as a sulphide, while the iron will remain in the solution.

Tardieu recommends to char the organs to be examined, by means of pure sulphuric acid and heat, in a porcelain capsule; the heat to be applied by means of a sand-bath, and urged until the bottom of the crucible is red-hot. The resulting carbonaceous mass is finely powdered after cooling, and treated with pure nitric acid, and evaporated at a gentle heat; the residue is diluted with water, and filtered; the filtrate evaporated to dryness, and heated, to expel all the nitric acid, and the residue dissolved in dilute nitric acid. Finally, sulphuretted hydrogen is transmitted through this solution, and the precipitate dried, treated with a few drops of nitro-muriatic acid, in a porcelain capsule, and evaporated to dryness. Solution of ammonia is now added, which will receive a blue coloration if copper be present, unless in excessively minute quantity: this solution is evaporated to dryness, and the residue treated with dilute hydrochloric acid. This last solution should respond to all the characteristic tests for copper.

In this connection it may be worth while to notice an observation of Tardieu (Sur l'Empoisonnement, p. 544) in relation to the employment of a porcelain crucible in preference to one of platinum, in the foregoing investigations. This author noticed that when beef's blood (and doubtless other animal matters) was incinerated in a *platinum* crucible, and treated with nitric acid, etc., and the resulting solution was brought in contact with a rod of polished iron, a reddish and somewhat brilliant deposit was made upon the iron, resembling copper in appearance. This does not occur if the experiment be performed in a *porcelain* capsule. M. Tardieu accounts for it by suggesting that probably the nitric acid, reacting upon the chlorides present in the animal matters, develops *aqua regia*, which dissolves a little of the platinum; and that the colored deposit on the iron is due to this metal. A natural inference from the above is, to avoid the use of platinum in this method of research for copper in the tissues; and likewise not to conclude that copper is present merely because the polished iron receives a reddish deposit from the suspected liquid.

Detection in the urine.—About six ounces of the suspected urine are evaporated to dryness; and the residue treated with strong nitric acid and chlorate of potash, with the aid of heat, to complete incineration. The resulting ash will contain the copper present, together with any iron: this is dissolved out by hot dilute nitric acid, and the liquid evaporated to dryness. The residue is dissolved in warm distilled water, and the solution tested in the usual way.

In relation to the question whether copper exists in the human body as a *normal* constituent, the weight of modern authorities is decidedly opposed to it. M. Tardieu informs us that his colleague, M. Roussin, experimented upon the body of a soldier suddenly killed in Algeria, with a view of determining the presence of normal copper in the tissues and organs, but with negative results (*loc. cit.*, p. 543). This metal does undoubtedly exist in minute quantities in the vegetable kingdom: its presence here has been ascribed to the soil in which the plants were grown. This would suggest the idea that it is an *accidental*, rather than a normal, constituent even of plants. At all events, the discovery of mere traces of copper in the human body after death ought never to be assumed as indicating copper-poisoning, unless the strongest evidence of this had been afforded by the symptoms, the morbid lesions, and the circumstances attending the case. As we have seen, copper may so easily and unknowingly be introduced into the system, through the food, and by other means, that an opinion of poisoning, based simply upon such slender grounds, would be altogether premature and unwarranted.

Quantitative estimate.—Use for this purpose the precipitated sulphide, which is to be dried and dissolved in hot, dilute nitric acid. To the boiling solution liquor potassæ is added as long as the oxide is precipitated, after which the boiling is continued for some time. After cooling, the precipitated black oxide is separated from the supernatant liquid by decantation, collected on a filter of known weight, thoroughly washed with warm water, and dried. It is then separated, as far as practicable, from the filter, and strongly ignited in a platinum capsule; and the ash of the filter, burned separately, is added to it; and, after cooling, the whole is weighed. Every 100 parts of the anhydrous protoxide represent 314.21 parts of pure crystallized sulphate of copper.

CHAPTER XVIII.

POISONING BY LEAD.

In its metallic state, lead appears to be destitute of poisonous properties. But as it is readily acted upon by the animal secretions and other matters found in the stomach and bowels, it undergoes oxidizement, when swallowed, and is converted into soluble, poisonous compounds. All the salts of this metal are more or less poisonous with perhaps, the single exception of the *sulphate*, which is very insoluble.

Cases of acute poisoning by the salts of lead are very rare: those that have occurred were chiefly due to accident. The *acetate* is one of the most active of these salts, and has been the most frequent cause of acute poisoning.

Slow or chronic lead-poisoning, on the other hand, is of very frequent occurrence. Of all the metals, none is so constantly and insidiously introduced into the human system as lead, under various forms: it even surpasses copper in this respect. In the arts, numerous classes of workmen are exposed to chronic lead-poisoning: thus, smelters of lead-ores inhale the fumes of the oxide; manufacturers of white and red lead receive it into their lungs in the dry powders of the carbonate and red oxide; the workmen who whiten Brussels lace by beating white lead into the fibre, constantly breathe an atmosphere of the poisonous carbonate, and often die from its effects; manufacturers of glazed cards suffer from the same cause. Painters, plumbers, pewterers, and glazers of pottery are all very much exposed to the same danger,—the former from inhaling the vapor of the oil of turpentine impregnated with the carbonate of lead; the latter, by the oxide employed in the glaze. Even sleeping in a freshly-painted room has been known to cause violent symptoms of colica pictonum, and even paralysis, in consequence, no doubt, of the volatile emanations containing the carbonate of lead.

Dr. Taylor (On Poisons, p. 434) alludes to himself as having suffered from severe colic though respiring the vapor of fresh paint. To show the frequency of this kind of poisoning, Dr. Clemens states that in ten years there were 1898 cases of chronic poisoning by lead among workmen admitted into the hospitals of Paris; and out of 1330 cases received in five years, 655 were among workers in white lead and painters. (Casper's Viertelj., 1853, ii. p. 177, quoted by Taylor.) The frequent handling of pewter vessels, and also of new type, has been known to produce lead-palsy: in the latter case the paralysis was purely local, being confined to the right hand. The sharp edges of the type caused abrasions of the thumb and fingers, a condition favorable to absorption. In the course of a week the paralysis was so complete that the hand dropped, and could not be voluntarily raised. There was also a faint blue line at the edge of the gums. (Jour. de Chimie, July, 1858, p. 434; quoted by Taylor.)

In domestic and culinary use, numerous cases occur of slow and accidental poisoning by lead. One of the most common examples is that afforded by the use of glazed pottery vessels. The inside glaze of these vessels is readily acted upon by ordinary acids, as vinegar, also by fats and oils, and likewise by alkalies. As vessels of this character are extensively used to contain various kinds of food, the oxide of the glaze is more or less dissolved, and is taken into the system along with the food. Dr. Taylor mentions an instance where *milk* was so much contaminated by being kept in glazed earthen pans, as to produce violent symptoms of colic, with vomiting, in four men who partook of it along with rhubarb-pie. In the matters vomited, lead was detected (*loc. cit.*, p. 447).

Cider and beer, if drawn through leaden pipes, acquire poisonous properties, in consequence of the formation of the malate or the carbonate of lead, which, although insoluble, may be diffused through the liquid and so be taken into the stomach. In some cases of the latter character, lead has been detected in the urine of the person using the beverage. Sometimes shot are employed to clean out wine-bottles, and, through carelessness, are not always removed before refilling the bottles with wine. In such cases the wine, even if of the better qualities (port and sherry), will after a time become more or less impregnated with the resulting salts of lead (principally the carbonate). So long as the wine is not agitated, this crust remains at the bottom, and the wine may be drunk with impunity. Domestic wines, such as those made from the currant, gooseberry, etc., contain much acid, and act readily upon the leaden shot in the bottles, speedily becoming contaminated with the poisonous salts of lead.

New rum is apt to contain lead, derived from the leaden worm of the still; while old rum, curiously enough, is free from this adulteration. Dr. Traill ascribes this difference, with great plausibility, to the fact that the old rum, being kept in oak casks, is deprived of its lead by the tannic acid contained in the oak, which precipitates it in an insoluble form. A most reprehensible practice formerly prevailed in France and England of adding litharge (protoxide of lead) to sour wines for the purpose of sweetening them: this proved to be a very prolific source of lead-poisoning. Even *distilled water* has been found to be impregnated with lead, when pipes of this metal have been used for condensing the vapor. In this way, also, such *aromatic oils* as are prepared by distilling the plants along with water, may become contaminated with lead.

Certain *medicinal substances* are frequently found to contain lead, which has been derived from their mode of manufacture. Thus, *carbonate of ammonia*, which is sublimed in leaden vessels; *borax* and other salts when crystallized in leaden pans; *tartaric acid*, according to M. Chevallier, from the employment of lead to sink the strings in the crystallizing solutions; and *acetic acid*, which, according to Dr. Taylor, has been found to contain as much as two per cent. of acetate of lead. Solutions of *potash* and *soda*, as is well known, when kept in flint-glass bottles, soon become contaminated with lead; and commercial *oil of vitriol* nearly always contains sulphate of lead, derived from the leaden chambers.

Various substances extensively used in the domestic economy are found to be occasionally contaminated with lead, such as flour, sugar, snuff, tobacco, chocolate, etc. A curious source of the adulteration of *flour* with lead was traced to the grinding of the wheat: a portion of the machinery had been stopped with lead cement and covered with plaster; the latter had given way, and the salt of lead, which fell out, had been ground up with the flour. (Jour. de Chim., 1857, p. 278.) A similar instance is mentioned in Taylor's Med. Jurisp. (Am. ed., 1873, p. 173): whole families, in one of the counties of the State of New York, in the year 1866, were poisoned by the use of flour manufactured at a mill the owner of which had been in the habit of filling the cavities of the millstones with lead. Refined *sugar* may contain traces of lead derived from the painted cones into which the syrup is poured. Snuff is often adulterated with red lead and chromate of lead to improve its color. Dr. Hassall detected out of forty-three samples of popular kinds of snuff, chromate of lead in nine, and oxide of lead in three samples (Food and its Adulterations, p. 591). The danger of using such snuff is very great, causing paralysis, wasting, and even death. (See Taylor on Poisons, p. 449.)

The practice of using an article termed *patent tin-foil* as a wrapper for tobacco, chocolate, bonbons, and farinaceous food for infants, has been attended with dangerous consequences. This foil is composed chiefly of lead, with a very thin outer coating of tin. When exposed to damp, this metallic alloy undergoes changes, resulting in the production of carbonate of lead, which impregnates the articles in contact with it. In France, the use of this spurious tin-foil has been interdicted, under a heavy penalty.

The *external* application of lead and its preparations is known to produce serious results. The effects of constant handling of pewter vessels and new type have already been alluded to. *Hair-dyes* and *cosmetics* notoriously contain lead, and have frequently occasioned paralysis, ophthalmia, and other unpleasant symptoms. A case is reported by Galtier, where a lead-plaster applied to an ulcer on the leg occasioned chronic lead-poisoning.

A case is reported in the British and Foreign Medico-Chirurgical Review (Oct., 1857, p. 525), where the external application of *white lead* to a scalded surface, as a dressing, produced unmistakable symptoms of lead-colic—such as acute abdominal pain, retraction of the umbilicus, constipation, and discoloration of the gums.

It is, however, chiefly through *drinking-water* that lead proves such a frequent and unsuspected cause of accidental poisoning. The conditions under which this result is brought about should be thoroughly understood by the medical jurist, as well as by the physician. It has been ascertained that absolutely pure water (containing no atmospheric air) has no action upon bright lead, if kept out of the air, in a hermetically-sealed tube; but the same water, if exposed to the air, soon deposits a milky film upon the surface of the metal, which in the course of twenty-four hours becomes a collection of pearly scales, either loosely adhering to the lead, or collected as a sediment at the bottom of the tube. This compound is a mixture of the hydrated oxide and the carbonate of lead. Although this compound is not soluble in the water, it is nevertheless diffused through it, and renders it highly poisonous for drinking purposes. *Rain* and *snow water* are the purest of all natural waters: they are destitute of saline ingredients, resembling in this respect artificially distilled water. Consequently, when such waters are collected from a leaden roof, or are kept in leaden cisterns, or are conveyed through leaden pipes, they soon become contaminated by lead, and acquire very poisonous properties. A notable instance of this is alluded to by Sir R. Christison (On Poisons, p. 526) as having occurred in Amsterdam. Previous to the use of lead instead of tiles for the roofs of the houses, lead-colic was of very rare occurrence; but after the introduction of leaden roofs the disease prevailed very extensively, and was of great violence, in consequence of the habitual use of the rain-water which fell from the roofs. Instances like the above are of very frequent occurrence, and they ought to serve as a warning against the dangerous practice of employing rain-water that has *in any way* been in contact with lead.

As regards the action of *spring* and *river waters* upon metallic lead, very much will depend upon the purity of the water,—*i.e.* the amount and the nature of its *saline* ingredients:

the purer the water, the greater the liability to contamination, and *vice versâ.* This will be better understood when it is remembered that the presence of certain salts in the water —particularly *sulphates*, *carbonates*, and *chlorides*—determines the speedy production of the corresponding salts of lead, which, being insoluble, form an incrustation upon the surface of the metal, that completely protects it from further chemical action, and thus acts as a preservative against future contamination by the metal. If the inside of a leaden pipe, through which spring or river water has been flowing for a long time, be inspected, it will be found coated over with a whitish incrustation composed of one or more of these salts of lead. The water supplied to Tunbridge, England, in the year 1815, was conveyed through leaden pipes, and produced an outbreak of lead-colic in that town. This water was found, on analysis, to be remarkably pure, containing only about one thirty-eight-thousandth part of its weight of solid ingredients, three-fourths of which were a feebly-protecting salt—the chloride of sodium. On the other hand, the waters which may be conveyed through leaden pipes without risk are such as contain a much larger proportion of saline constituents,—sufficient to produce the deposit on the surface, just mentioned. This is, fortunately, the case with the great majority of rivers and springs: hence the immunity from danger to those using such waters, although these are conveyed to their houses through lead pipes. According to Christison, the water of Edinburgh, although containing but about one twelve-thousandth part of solid matter, is almost entirely protected from the action of lead. The river Thames contains about seventeen grains of saline matter to the gallon, and is remarkably free from any action of lead. The Claremont water, which contains only five grains of salines to the gallon (of which one-half is chloride of sodium), produced a very severe form of lead-colic in the course of a few months, in a number of persons who drank them. The water of the river Schuylkill, which chiefly supplies the city of Philadelphia, contains from six to eight grains of saline matters to the gallon: it has never been known to produce lead-poisoning, although conveyed through leaden pipes.

The salines which are most protective to the water are the *sulphates;* next to these, the *carbonates;* and last, the *chlorides.* It has been found that while certain waters may be preserved in leaden cisterns with impunity, if these be covered with leaden lids the latter will become coated with an incrustation of carbonate of lead, arising from the contact of the vapor of water (which may be considered as distilled water) with the surface of the metal. This may in time fall into the cistern, and so contaminate the water. It is quite possible that some instances of rapid corrosion of lead in contact with water containing salines, are due to a *galvanic* action. The presence of small portions of other metals in the lead would be quite sufficient to set up such an action; as, for example, where sheets of lead are connected together by solder.

Acetate of Lead.—Sugar of Lead.—This salt may be taken as the type of the preparations of lead which occasion acute poisoning. It commonly occurs in heavy crystalline masses; white, or nearly so; somewhat resembling loaf-sugar in appearance, for which it has been mistaken. It has an acetous odor, and a sweetish, astringent taste; it is very soluble in water, producing with ordinary water a milky solution, owing to the carbonic acid contained. It is less soluble in alcohol.

Sugar of lead cannot be regarded as a very active poison. It is frequently given in medical practice to the extent of twenty grains, or more, daily, without injurious effects; although the continuation of such quantities for many days would be likely to bring on the specific action of lead. In doses of one or two ounces the symptoms are as follows: a burning and pricking pain in the throat and gullet, thirst, vomiting, colic pains, with tenderness of the abdomen, and obstinate constipation; retraction of the walls of the abdomen; cramps; cold sweats; and, in fatal cases, convulsions and tetanic spasms. The urinary secretion is generally diminished. Occasionally there is a variation in some of the above symptoms: thus, in some instances there is severe and bloody purging; again, the discharges from the bowels are hard, dry, and of a black color. The pulse is usually slow and feeble, but sometimes quickened. The intellect generally remains clear. Should the case be protracted, there would likely be cramps in the legs, pain in the insides of the thighs, numbness, and sometimes paralysis of the limbs. Other acute nervous symptoms occasionally observed are giddiness, torpor, and even coma. The peculiar blue line upon the gums, which is so generally attendant upon chronic lead-poisoning, may also be sometimes noticed in acute cases.

Fatal dose.—In the few fatal cases reported, the quantity taken could not be accurately determined. Instances are mentioned where an ounce was swallowed with impunity; but, as a rule, this quantity would be very apt to be followed by dangerous, if not fatal, consequences.

Fatal period.—Dr. Taylor mentions two cases where a solution of the subacetate (Goulard's extract) taken by two children respectively, in unknown doses, destroyed life within thirty-six hours. The symptoms, at first, resembled those of Asiatic cholera,—there being violent vomiting and purging. (On Poisons, p. 430.) Dr. Beck refers to a case of a soldier who swallowed an unknown quantity of sugar of lead in solution: he was soon seized with violent symptoms, indicating gastro-enteric irritation, and died in great agony at the end of three days. Another case is quoted by Sir R. Christison (On Poisons, p. 430): an unknown quantity of Goulard's extract was swallowed by a soldier; on the second day he was affected with loss of appetite, paleness, costiveness, and extreme debility; on the third day he had severe colic, retraction of the abdomen, loss of voice, cold sweats, trismus, and convulsions; he died before the close of the same day.

Treatment.—In cases of acute poisoning by acetate of lead, free emesis should be excited by sulphate of zinc, which, besides evacuating the stomach, acts antidotally by converting the poison into the insoluble sulphate of lead. This may be followed by copious draughts of milk and white of egg, as both casein and albumen form insoluble compounds with oxide of lead. The stomach-pump may be occasionally employed with benefit. Sulphate of magnesia, or sulphate of soda, should then be administered, with the twofold purpose of acting as an antidote and as a cathartic. Castor oil and croton oil have also been recommended as purgatives. M. Bouchardat has proposed the *hydrated persulphide of iron* as a good chemical antidote. The urine should be frequently examined, for the purpose of tracing the disappearance of the poison from the body.

Post-mortem appearances.—According to Dr. Taylor, the lesions observed in cases of acute lead-poisoning are very characteristic. The mucous lining of the stomach and bowels is covered with a thick white or whitish-yellow layer of mucus mixed with a salt of lead, beneath which the membrane is reddened, or ecchymosed. In some cases this membrane has been found abraded in several places, particularly near the pylorus, and other parts of the stomach have been in a state of high inflammation. It appears that it is the neutral salt alone which acts as a corrosive, this effect not being manifested when the acetate is combined with an acid. The intestines are sometimes much contracted. It should not be forgotten that acute poisoning by lead may result fatally, without leaving behind any very noticeable pathological changes.

CHRONIC POISONING.—*Symptoms.*—Deleterious effects are more frequently observed from the slow and insidious introduction of lead into the system, than in the case of any other metal. Chronic poisoning may result from any of the preparations of lead, but is most commonly traceable to the carbonate (white lead) and litharge, in the artisans connected with lead-works, or to the accidental impregnation of drinking-water and other beverages, or of articles of food. These effects first show themselves in *lead colic*, and subsequently in *lead palsy*.

Lead colic (*colica pictonum*, or *painter's colic*) is characterized by excruciating pain in the abdomen around the umbilicus, which is generally relieved by pressure. The abdomen is hard, its muscles strongly contracted, and its walls more or less drawn inwards; the bowels are usually obstinately constipated, though often attended with a feeling of desire for their evacuation. Scanty evacuations are sometimes passed, with much suffering. The urine is scanty, and voided with difficulty. The countenance is dull and anxious; the skin bedewed with cold perspiration; the pulse either about natural, or else accelerated; the breathing quick and catching; fever is very rare; there is loss of appetite, and dryness of the mouth and throat. The skin is dry and icterode, and there is often a metallic or astringent taste in the mouth. The breath is fetid. The *blue* or *saturnine line* at the edge of the gums, first pointed out by Dr. Burton, is nearly always observed in cases of chronic lead-poisoning. This discoloration is ascribed, with great plausibility, to the elimination of the poison in the buccal secretions, in the form of the dark-colored sulphuret. It is said to be most marked around the upper incisors. Although this blue line is a valuable indication of lead-poisoning, it should be remembered that it occasionally occurs as the result of the introduction into the system of other metals, as mercury and silver. Moreover, cases of undoubted poisoning by lead do occur where this sign is altogether wanting: so that, while its presence may generally be regarded as indicative of lead-poisoning, its absence is not to be taken as a proof that this poison is not in the system. In some cases in which the blue line has been absent, the gums have presented a fungous appearance, and have bled very easily. (Med. Times and Gaz., Jan. 30, 1858.)

The earliest period at which this blue line on the gums first shows itself is not ascertained. Dr. Burton states that he has seen it produced in twenty-four hours after giving four doses, of five grains each, of acetate of lead; and he thinks that it would occur still earlier, if larger doses were taken. When it is once established, it is very persistent. Instances are mentioned of its presence being still visible *four years* after exposure to the source of poisoning had ceased. (Med. Times and Gaz., 1848, p. 195; quoted by Taylor.)

Lead-colic may terminate in recovery, or it may pass on to the second form of chronic poisoning—*lead-palsy*. This is sometimes the termination of a single attack of colic; but more commonly it supervenes after repeated seizures. Again, it may come on without any previous attack of colic. It affects chiefly the upper extremities. There is first a dull, numb feeling in the skin, especially of the fingers and forearms; trembling of the arms and legs, unsteadiness in walking, loss of power in the hands and arms, which gradually waste away. This loss of muscular power is chiefly confined to the extensors of the hand, so that when the arm is raised the hand drops by its own weight: whence the common expression "wrist-drop," or "hand-drop." Symptoms of brain-affection sometimes present themselves, such as giddiness, torpor, and apoplexy. In cases which pursue a slow course to death, the paralysis may gradually extend to all the muscles; epileptic paroxysms occur at intervals, and there is general œdema, with whitened skin, indicating the increasing anæmia. Sometimes the case is further complicated with albuminuria; locomotion becomes impossible, and the patient dies in convulsions or coma, or from paralysis of the respiratory muscles. After death, lead has been found in the tissues, especially in the gray matter of the spinal cord. (Comptes-Rendus de la Soc. de Biol., iv. 1862; quoted by Dr. H. C. Wood in his "Therapeutics," 1874.)

Workers in white-lead factories are particularly exposed to the danger of slow poisoning by lead, more especially if the carbonate be ground in the dry state, in which condition it is diffused through the atmosphere and freely respired into the lungs. It is also introduced through the skin, adhering to the cutaneous secretions, owing to the general absence of cleanliness on the part of the workmen. Since the practice of grinding the white lead under water has prevailed, cases of colica pictonum arising from this source have very sensibly diminished in number. The best practical means of avoiding the danger is the strict enforcement of cleanliness, especially before eating, and the habitual use, as a beverage, of very dilute sulphuric acid.

There is no doubt that obscure cases of supposed spinal, cerebral, or heart disease are really due to the unsuspected and insidious introduction of lead into the system. In such cases, a close and critical examination should be instituted into the employment, mode of living, and particularly the source and mode of conveyance of the drinking-water used by the patient.

Chemical analysis.—1. *In the solid state.*—The acetate, when heated on a piece of porcelain, first fuses, and is reduced to a white, anhydrous mass: if the heat be continued, the mass again fuses, and then becomes dry and charred, slowly assuming a reddish-brown color, and consisting of a variable mixture of the oxides of lead. The *carbonate* treated in the same manner does not fuse, but is converted into a similar colored mixture of the oxides. A fragment of the acetate placed on charcoal, and exposed to the inverse flame of the blowpipe, is decomposed, with the production of bright malleable globules of metallic lead, and a surrounding yellow incrustation of the oxide. A fragment of acetate of lead dropped into a solution of *iodide of potassium* assumes a bright yellow color, due to the formation of the iodide of lead. This is soluble in an excess of the reagent. This is a very delicate test: even one ten-thousandth of a grain, if deposited on one point of the iodide of potassium solution, gives the yellow hue (Wormley).

If the same experiment be made in a solution of *bichromate of potassa*, it likewise assumes a yellow tint, from the formation of chromate of lead. Sulphuretted hydrogen, or sulphide of ammonium, immediately imparts a black color to a fragment of the acetate. If the powder be boiled in a tube with diluted sulphuric acid, acetic acid, recognized by its odor and volatility, escapes.

2. *In the liquid state.*—(1) A few drops of the solution of the acetate allowed to evaporate spontaneously on glass will crystallize in opaque needles, which are colored yellow when touched with a drop of solution of iodide of potassium, or chromate (or bichromate) of potassa; and black, by sulphide of ammonium. (2) Dilute sulphuric acid produces a copious white precipitate (sulphate), soluble in hydrochloric acid, and in a large excess of caustic potassa. If the lead solution be very dilute, the precipitated sulphate does not separate until after some time. (3) It is precipitated bright yellow by *iodide of potassium:* the precipitated iodide of lead is soluble in caustic potash, and in concentrated hydrochloric acid. It is also soluble in boiling water, which deposits it in brilliant yellow six-sided tables, on cooling. This is an excellent and

reliable test. (4) *Bichromate of potash* also precipitates it as yellow chromate of lead: this is readily soluble in potash, but only slowly soluble in hydrochloric acid, which converts it into white chloride of lead. (5) *Sulphuretted hydrogen* is the most delicate of all the tests. According to Dr. Taylor, a current of this gas when properly applied will reveal a quarter of a grain of a salt of lead in a gallon of water, or about one three-hundred-thousandth part. The color of the precipitated sulphuret of lead is black; but as there are other metals, some of whose salts give with sulphuretted hydrogen or with sulphide of ammonium black precipitates, such as mercury, silver, copper, cobalt, nickel, bismuth, tin, and iron, the suspected sulphide must be subjected to a further proof before deciding that it contains any lead. Its true character may be established by placing a fragment on a piece of charcoal and applying the inner flame of the blowpipe upon it: a metallic globule will be obtained, possessing all the characters of lead. Or, the precipitate may be dissolved in dilute nitric acid with the aid of heat, evaporating to dryness, dissolving in water, and applying the usual tests for the liquid form of lead compounds.

The precipitated sulphide of lead is insoluble in dilute mineral acids, and also in the caustic alkalies. It is soluble in hot hydrochloric and nitric acids, forming, in the first instance, white chloride of lead, which, unless it be in very minute quantities, separates, as the liquid cools, in the form of beautiful crystalline plates. With hot nitric acid it forms a nitrate of lead with the separation of *free sulphur.* If the acid be concentrated and the heat be continued, the free sulphur becomes oxidized to sulphuric acid, which displaces the nitric acid, and unites with the oxide of lead to form a sulphide.

(6) *The zinc or galvanic test.*—This is a very delicate and satisfactory test. A drop or two of the suspected solution, acidified with acetic acid, is placed upon clean platinum-foil, and a thin, polished strip of zinc is made to touch the platinum, through the liquid: crystals of metallic lead are instantly deposited on the zinc. Or, a drop of the fluid is put into a watch-glass, and a fragment of zinc is dropped into

it: very soon metallic lead is precipitated upon the zinc in a beautiful arborescent form (*lead tree*), which should be viewed under the microscope at once, before any deposition of the carbonate occurs. A salt of tin, under similar circumstances, will also give an arborescent precipitate of metallic tin. Hence the true nature of the *metal* thus obtained must be verified by converting it into a salt by nitric acid, and then applying the usual tests.

Several other tests are noticed in the books, such as *potassa* and *ammonia*, the *alkaline carbonates*, *oxalate of ammonia*, *yellow and red prussiate* of potassa; but, as these are not characteristic, and are inferior in value to those already mentioned, they need not be here detailed.

Detection in organic matters; and in the contents of the stomach.—As acetate of lead is readily decomposed by various organic substances, such as albumen, mucus, and tannin, which precipitate the oxide of lead, the mixture presented for examination may contain the poison in either the solid or the liquid portion, or in both. As a trial test, it may be proper first to filter off a portion of the liquid, and test it with sulphuric acid; or with sulphuretted hydrogen, by exposing a piece of bibulous paper dipped into the liquid to a current of this gas: a brown stain upon the paper would indicate the presence of lead, though not of this metal *exclusively*. If the paper is not stained brown, no perceptible quantity of lead can be present.

Supposing the trial test indicates the presence of lead; the mixture should be acidulated with pure nitric acid and boiled for some time: this will dissolve to a great extent the organic compounds of lead. After cooling, it should be filtered, and the solids on the filter thoroughly washed and reserved for future examination. The filtrate should now be concentrated by evaporation, and a stream of washed sulphuretted hydrogen gas passed through it, to the point of saturation. After standing for some time in a warm place, the precipitate is collected on a filter, from which it is carefully washed into a test-tube or capsule, by a jet of water. After the precipitate has subsided, the supernatant water may be removed by decantation, a small quantity of nitric acid added, and a gentle heat applied. By this means the sulphide is converted into the soluble nitrate, with the separation of free sulphur. A small quantity of distilled water is now added, the mixture filtered, and the filtrate subjected to the usual tests for lead, as already pointed out. Should the quantity of the liquid be too small for the application of all the tests, it would be best first to employ sulphuric acid: if this agent cause a white precipitate, soluble in potassa (proved to be free from lead), and this solution be turned black by sulphide of ammonium, the presence of lead may be regarded as established. According to Prof. Wormley (*loc. cit.*, p. 370), the one-thousandth of a grain of oxide of lead diffused through ten grains of water, and precipitated as sulphide, when treated with one drop of nitric acid, will yield a clear solution which gives very visible reactions with the appropriate tests for lead.

In case no lead is found dissolved in the liquid portion examined, the solids reserved should be boiled for some time with water containing about a fourth part of nitric acid; the cooled solution filtered; the filtrate evaporated to dryness, and incinerated, in order to destroy the organic matter. The residue is to be dissolved in a little nitric acid, and properly diluted for testing.

In all the above reactions, care must be taken to expel the whole of the nitric acid (or else carefully to neutralize it by pure potassa), as an excess of the acid will show a yellow color with the iodide of potassium test, although no lead be present.

The *contents of the stomach* should be examined as above, by boiling with dilute nitric acid, etc. If dilute sulphuric acid, or an alkaline sulphate, had been administered as an antidote, the poison would probably be found in the form of an insoluble white sulphate of lead, adhering to the coats of the organ. Under such circumstances, the suspected substance should be carefully scraped off, and boiled in a strong solution of pure potassa (which dissolves it), and the lead precipitated by sulphuretted hydrogen.

From the tissues.—The solid organ,—the liver, for example,—in sufficient quantity, is cut into fine pieces and boiled in a mixture of one part of pure nitric acid to four parts of distilled water until the mixture becomes homogeneous. It has been recommended by some authorities to employ chlorate of potassa along with the nitric acid, with a cautious application of heat, until the disappearance of all vapors. When the mixture has cooled, it is to be filtered, and the filtrate evaporated to dryness; the residue moistened with nitric acid, and again evaporated to dryness; and the heat cautiously increased until all vapors cease to be given off, and the residue is reduced to a carbonaceous mass. This mass is powdered and boiled with a small quantity of strong nitric acid diluted with water, the solution filtered, the filtrate evaporated to dryness, and the residue dissolved in a little water acidulated with nitric acid. This solution, filtered if necessary, is saturated with sulphuretted hydrogen; the precipitated sulphide, after being washed, is boiled in dilute nitric acid; and the resulting solution subjected to the usual tests.

M. Tardieu recommends, among other methods of destroying the organic matters, carbonization by means of concentrated sulphuric acid and heat. In this case, however, it must be remembered that the greater portion, if not all, of the lead will be found in the state of sulphate, mixed up with the carbonaceous mass. This mass is to be finely powdered, and boiled for one hour with a solution of carbonate of soda or potassa. The black-colored mixture is then filtered; the solids on the filter are first thoroughly washed with pure water until the washings cease to be alkaline, and then frequently with very dilute nitric acid, for the purpose of dissolving the carbonate of lead formed. The acid liquids being mixed together are then saturated with sulphuretted hydrogen; and after standing for several hours, the precipitated sulphide is treated in the manner already described (*loc. cit.*, p. 737).

Still another method of treating the solid organs is, first to dry them thoroughly, and then to incinerate them in a porcelain crucible, and dissolve out the lead by means of strong nitric acid; evaporate the residue to dryness; dilute with water, and precipitate by sulphuretted hydrogen. In case of the detection of minute portions in the organs of a body suspected of being poisoned, a careful inquiry should always be instituted in reference to the particular occupation, mode of living, etc., of the individual; remembering how insidiously and unsuspiciously lead may be introduced into the system, and that, too, many months before death.

The urine.—As it is chiefly through this secretion that lead is eliminated from the system, the examination of the urine ought never to be neglected, especially in obscure cases of suspected lead-poisoning. For the purpose of analysis, fifteen or twenty ounces of urine, acidulated with nitric acid, should be evaporated to dryness, the residue carbonized with nitric acid, and the carbonaceous mass treated in the manner directed for the detection of lead in the tissues (p. 319).

Quantitative determination.—Lead is usually determined as a sulphide; which is precipitated from a slightly acid solution by a slow stream of washed sulphuretted hydrogen in a warm place, until the whole of the precipitate has subsided. This is then collected on a filter of known weight; thoroughly washed, dried until it ceases to lose weight, and weighed.

Every 100 parts of the dry sulphuret represent 93.31 parts of oxide of lead, or 158.37 parts of crystals of the acetate.

Every 100 parts by weight of dry sulphate of lead are equivalent to 125 parts of crystallized acetate.

CHAPTER XIX.

POISONING BY ZINC, BISMUTH, TIN, IRON, AND CHROMIUM.

SECTION I.

POISONING BY ZINC.

CASES of poisoning by the preparations of Zinc are rare, except from accident. Although *metallic* zinc is harmless so long as it retains its metallic condition, it would nevertheless be likely to occasion serious results if swallowed, in consequence of its easy conversion into soluble salts from contact with the contents of the stomach. The *sulphate* and the *chloride* are the preparations of zinc that are the most common causes of poisoning.

The zinc of commerce (spelter) very often contains *arsenic*, *cadmium*, *antimony*, *iron*, *lead*, and other impurities.

SULPHATE OF ZINC (*White Vitriol*).—This salt usually occurs in white, prismatic crystals. It has a metallic, astringent taste; is very soluble in water; insoluble in alcohol and ether; and effloresces on exposure to the air.

Effects on the system.—Sulphate of zinc is a very prompt and powerful emetic, and is much used for this purpose in cases of narcotic poisoning. It has been given medicinally in quite large doses for a considerable length of time, without producing any injurious effects. The late Dr. Babington administered it, in cases of epilepsy, to the extent of two scruples three times a day for a period of three weeks, without even occasioning any symptoms of irritation. The medicine was first given in small doses, gradually increased. A tolerance of the medicine seemed to have been established, similar to that attending the exhibition of antimony as administered on the *contra-stimulant* plan.

When swallowed in doses of half an ounce to an ounce, the effects are those of a powerful irritant poison. A strong metallic taste is perceived, attended with a sense of burning and constriction of the throat and gullet; nausea; violent vomiting and retching; intense pain of the stomach and bowels; frequent purging; small and frequent pulse; great anxiety; cold perspiration; extreme prostration, and death. The intellect is generally unaffected to the last.

Dr. Taylor mentions the case of a man who recovered in a few days after taking an ounce of sulphate of zinc by mistake for Epsom salts. There were early vomiting and purging of a violent character, with great prostration of strength. (Med. Jurisp., Am. ed., 1873, p. 183.) A fatal case is quoted by Prof. Wormley from the "Am. Jour. of Med. Sci.," July, 1849,—that of a woman who swallowed, by mistake for Epsom salt, a solution containing an ounce and a half of sulphate of zinc. Death ensued in *thirteen hours and a half*, after most violent vomiting and purging, and pain in the abdomen and the limbs, together with extreme prostration and anxiety, and small and frequent pulse. A sister of this woman, aged thirty-five years, took a similar dose of the poison, but recovered, after suffering from its effects for several days.

Morbid appearances.—These vary somewhat in different cases. Sometimes there is simply inflammation of the stomach, in patches. In other cases there are evidences of the most violent irritation and inflammation, such as a softened, gelatinous condition of the mucous membrane, which is easily scraped off; ecchymosed patches, and sometimes ulceration, together with injection of the small intestines; a yellowish, pultaceous matter covering the inner surface of the stomach and bowels; congestion of the brain and its membranes; bloody effusion into the pleura; congestion of the lungs, and a flaccid condition of the heart. The above lesions do not all occur in the same cases; but they represent the general post-mortem appearances observed.

CHLORIDE OF ZINC, in the liquid form, is sold in the shops under the name of "Sir Wm. Burnett's Disinfecting Fluid." It is much used as a deodorizer. It contains about two hundred grains of the anhydrous salt in each fluidounce. It is a powerfully corrosive fluid, and has been frequently the cause of death, being taken either by mistake, or suicidally.

The *symptoms* of poisoning by this substance are, in general, similar to those produced by the sulphate, only more intense in their character, and resembling somewhat those of the corrosive acids. These violent symptoms come on *immediately* on swallowing the liquid; the matters vomited and purged are frequently tinged with blood and mixed with shreds of mucous membrane. There has been observed frothing at the mouth, with a white appearance of the inside of this cavity.

The period at which the chloride of zinc proves fatal varies, as in poisoning from other substances. Dr. Taylor records the most rapidly-fatal case known,—that of a woman aged twenty-eight years, who swallowed an ounce of this fluid and survived only *four hours.* On the other hand, the case may become chronic, and the patient perish at last, after months or years of suffering, from stricture of the œsophagus, or from the resulting exhaustion and emaciation.

The *post-mortem appearances* in poisoning by the chloride of zinc are those of a corrosive as well as of an irritant. In some cases the coats of the stomach have been found hard and leathery, thickened and corrugated; in others, the stomach was reddened externally, the mucous membrane of a deep-purple color, and partially corroded and destroyed. The pyloric opening was constricted, and its mucous membrane appeared as if it had been cauterized. Constriction of the œsophagus has been noticed; together with a pseudo-membranous deposit on the lining membrane. The *lungs* have been found congested, as also the vessels at the base of the brain. The *heart* is usually unaffected. In very protracted cases, the stomach has been found so much contracted as to contain only four ounces of fluid; with one or more perforations.

Treatment.—Free emesis should be encouraged by the copious use of warm, mild, mucilaginous drinks: the stomach-pump may sometimes be advantageously used. Albumen should be freely given. The excessive irritation is best combated by opium.

Chemical analysis.—In the solid state, the sulphate may be distinguished from Epsom salt and oxalic acid (which it closely resembles in appearance) by the action of chromate of potassa: a solution of the latter applied to a grain or two of the sulphate, in a watch-glass, changes it to a yellow color, and it soon becomes converted into a mass of small, yellow granules.

The blowpipe affords an easy method of identifying a salt of zinc. A small fragment, previously mixed with a little carbonate of soda, is placed on a piece of charcoal, and the inner flame made to play upon it. It quickly fuses, and is soon dissipated into vapor of the oxide, which forms a yellowish incrustation upon the charcoal, which on cooling becomes white. Under the blowpipe cobalt imparts a *green* color to the fused bead of zinc.

In solution: (1) The fixed alkalies and ammonia throw down a white, *hydrated oxide of zinc*, soluble in excess of the precipitant. (2) The alkaline carbonates precipitate the white hydrated carbonate, insoluble in excess of the precipitate, but soluble in excess of carbonate of ammonia. (3) Ferrocyanide of potassium gives a *white* precipitate, insoluble in acids and alkalies. (4) Sulphuretted hydrogen, or sulphide of ammonium, occasions a white, amorphous precipitate (sulphide of zinc) in neutral or alkaline solutions, insoluble in the alkalies and organic acids, but readily soluble in hydrochloric acid. The precipitate is *white* only if the zinc be perfectly free from iron and other impurities. The white precipitate should always be further verified, inasmuch as a whitish deposit, consisting chiefly of free sulphur, may be thrown down from other solutions by sulphuretted hydrogen and sulphide of ammonium. The suspected sulphide of zinc should be collected on a filter, boiled in hydrochloric acid, the solution filtered and diluted, and subjected to the usual liquid tests.

Other tests may be employed, such as *carbonate of potassa*, *phosphate of soda*, and *oxalic acid;* but they are not characteristic, and are of inferior value to those before mentioned.

The *acids* in the compounds may be recognized by their appropriate tests: *sulphuric acid* by chloride or nitrate of barium; *hydrochloric acid*, by nitrate of silver.

Detection in organic mixtures.—The contents of the stomach, or other organic mixture supposed to contain the poison, should be mixed with a little acetic acid, and heated gently for some time, in order to dissolve out the zinc that may have combined with any albumen, fibrin, or casein. After cooling, the solution is to be filtered, concentrated, if necessary, and then treated with sulphide of ammonium, or sulphuretted hydrogen, as long as any precipitate is thrown down. The latter is collected on a filter, washed, and digested in nitric acid: this converts it into a nitrate. It is now to be evaporated to dryness, in order to expel the excess of acid; the residue dissolved in distilled water, and the filtered liquid examined by the ordinary zinc tests. As the preparations of zinc generally contain *iron*, the presence of the latter metal may more or less modify the chemical reactions of the former. The iron may be separated by adding an excess of ammonia, which precipitates the oxide of iron, whilst it retains the zinc-oxide in solution. After filtration, the zinc may be precipitated by sulphuretted hydrogen.

Detection in the tissues.—Absorbed zinc may be procured from the tissues or organs by reducing them to small fragments, and boiling in nitric acid somewhat diluted, until all the organic matter is thoroughly dissolved. When cold, the solution is strained, and the liquid evaporated to dryness. Pure nitric acid is sprinkled over the residue, which is heated until the organic matter is completely destroyed. The dry mass thus obtained is treated with distilled water containing a little hydrochloric acid; and the filtered liquid evaporated to dryness. The residue is dissolved in distilled water, and precipitated with sulphide of ammonium: the precipitate should be identified in the manner already pointed out. Or, the viscera, after being thoroughly dried, may be incinerated in a porcelain crucible, and the resulting ash treated with nitric acid; the solution filtered and evaporated to dryness; the residue dissolved in water acidulated with hydrochloric acid; again evaporated to dryness, and then dissolved in pure water, and tested by sulphide of ammonium, as just described.

Inasmuch as *chloride of zinc* is frequently used for embalming and preserving dead bodies, the discovery of zinc in the tissues in a suspected case is no evidence of the death having resulted from this poison, unless supported by other proofs.

Absorbed zinc has been detected in the tissues and in the blood after death, after comparatively long periods.

Quantitative determination.—Zinc is generally determined as an oxide. The solution is heated to the boiling temperature, and precipitated with a dilute solution of carbonate of soda until all the precipitated carbonate subsides. This should then be collected in a filter, washed with hot water, dried, and ignited. The protoxide of zinc thus obtained is now weighed. Every 100 grains represent 354.13 grains of pure crystallized sulphate, or 167.77 grains of anhydrous chloride of zinc.

SECTION II.

POISONING BY BISMUTH.

Subnitrate of bismuth.—Pearl white.—Magistery of bismuth.—This substance is considerably employed both as a cosmetic and as a medicine. For the latter purpose it is frequently administered in doses of five to thirty grains, in certain derangements of the stomach and bowels. Several fatal cases have been reported as resulting from large doses of this substance: Dr. Taylor records one in which *two drachms* produced death in an adult in nine days. The symptoms were those of the powerful irritant mineral poisons,—identical, indeed, with the symptoms commonly seen in arsenical poisoning. This authority states (Prin. and Prac. of Med. Jurisp., 1873) that the medicinal subnitrate generally contains arsenic as an impurity. He detected it, in a comparatively large proportion, in samples obtained from three respectable London druggists. Three specimens out of five contained it. The arsenic may readily be discovered by dissolving the subnitrate in pure hydrochloric acid, slightly diluted, and using a Marsh's apparatus. The same adulteration has been occasionally found to exist in the subnitrate of bismuth sold in our own shops; and it should be looked to by physicians, as being the probable cause of the irritation which occasionally follows the use of this medicine.

Dr. Fullerton, of Hillsborough, Ohio, relates an instance of poisoning by impure subnitrate of bismuth. A physician having occasion to put himself under this remedy noticed in a few days a puffiness of his eyelids and some gastro-intestinal irritation, which symptoms disappeared on discontinuing the medicine, but were again manifested on its renewal. On analysis, a large proportion of arsenic was detected in the subnitrate. (Am. Jour. Med. Sci., Jan., 1874.) The editor of the above journal also quotes a case of an infant, recorded by Dr. Herbert in "Le Mouvement Médical," Nov. 22, 1873, where the irritating symptoms resulting from the employment of subnitrate of bismuth for a diarrhœa were traced to the adulteration of the medicine by arsenic.

This impurity may essentially modify a medico-legal opinion as to the presence of traces of arsenic in a body, where bismuth had been previously administered medicinally. An interesting case of this nature (State of Virginia *v.* Mrs. E. E. Lloyd, 1872) was recently tried, in which the defense strongly contended that the existence of a fractional portion of arsenic, alleged to have been found in the liver of the deceased, was to be ascribed to the subnitrate of bismuth which had been taken before death: this bismuth was afterwards found to be contaminated with arsenic. The prisoner was acquitted.

By the process recommended by the present U. S. Pharmacopœia, the bismuth is entirely freed from arsenic.

Subnitrate of bismuth occurs in the form of a white powder, insoluble in water, but soluble in nitric acid; the solution, when thrown into water, yielding a copious white precipitate. It is blackened by sulphuretted hydrogen and by sulphide of ammonium.

A piece of paper wetted with a solution of sulpho-cyanide of potassium, and dried, is a very sensitive and characteristic test for a soluble salt of bismuth,—a beautiful yellow spot appearing at the point of contact. According to MM. Bergeret and Mayençon, after the administration of the subnitrate the metal may thus be always detected, after a few hours, in the urine. (Journal de l'Anatomie, 1873, p. 242; quoted in Dr. H. C. Wood's "Therapeutics.")

SECTION III.

POISONING BY TIN, SALTS OF IRON, AND CHROMIUM.

Salts of Tin. — The only preparations of tin requiring notice are the *chlorides*. A mixture of the protochloride and perchloride, in solution, constitutes what is sold under the name of *Dyers' Spirit*.

The effects of these salts upon the system are those usually attendant upon the mineral irritants. Cases of poisoning from them are very rare.

The *protochloride of tin* is distinguished by the following properties: (1) It is precipitated of a dark chocolate color by sulphuretted hydrogen; also by sulphide of ammonium,—the precipitate being soluble in an excess of the reagent. (2) Bichloride of mercury gives a gray precipitate of metallic mercury. (3) Chloride of gold gives a fine purple precipitate,—the purple of Cassius. (4) A fragment of zinc immediately precipitates metallic tin in a beautiful arborescent form, very much as in the case of lead.

The *perchloride* is precipitated yellow by sulphuretted hydrogen and sulphide of ammonium, the precipitate being soluble in an excess of the reagent. This yellow precipitate is distinguished from the yellow sulphide of arsenic by being insoluble in ammonia; and from sulphide of cadmium, by being insoluble in hydrochloric acid. Corrosive sublimate and chloride of gold give no precipitate with it.

The preparations of *silver*, *gold*, and *platinum* are highly irritant and corrosive; but they so rarely occasion poisoning in the human subject, that further notice of them is unnecessary.

Preparations of Iron.—The only salts of iron necessary to mention as possessing poisonous properties are the *sulphate* (*green vitriol*) and the *chloride*.

The sulphate, in large doses, is a powerful irritant, and has proved fatal in several cases recorded by Sir R. Christison, Orfila, and others.

The chloride, in the form of *tincture* (*muriated tincture of iron*), is much used in medicine; but in large doses it is a powerful irritant and corrosive, giving rise to symptoms very like those produced by the mineral acids, such as dryness and swelling of the throat, burning pain in the stomach, vomiting of blood, and black evacuations from the bowels. Dr. Christison relates a case of a man who swallowed, by mistake, an ounce and a half of this liquid: death occurred in about five weeks. The stomach was found inflamed, and thickened towards the pyloric orifice.

It may be well to remember that this substance is sometimes employed by pregnant women as an abortive.

Preparations of Chromium.—Two salts of chrome—the neutral chromate and the bichromate of potassa—are manufactured extensively, and are much used as dyes.

The *chromate of potash* is of a yellow color. The *bichromate of potash* occurs in beautiful orange-red crystals, in the form of rhombic plates or prisms, very soluble in water. This salt is a powerful irritant, and has proved fatal in several instances. In one case, communicated to Dr. Taylor by Mr. Wood, of St. Bartholomew's Hospital, two drachms killed a woman in four hours, with symptoms of violent irritation: the post-mortem appearances were those of a corrosive poison. Mr. Wilson, of Leeds, mentions a fatal case in which there was an entire absence of vomiting and purging (Med. Gaz., vol. xxxiii. p. 734). In this case the salt appeared to have acted not so much by its irritant properties, as by the indirect effect upon the nervous centres. Certain kinds of ink-powder, composed of this salt, have been known to produce injurious, and even fatal, effects.

Workmen engaged in the manufacture of the bichromate are often exposed to its noxious influences. Several fatal cases have occurred in Baltimore, where it is largely manufactured. Dr. Baer, of that city, reported a case of a laborer who, attempting to draw off from a receiver a solution of this salt, accidentally imbibed, through the siphon, a small quantity into his mouth. In a few minutes he experienced great heat in the throat and stomach, which was followed by violent vomiting of blood and mucus. The vomiting continued incessantly till his death, which took place in *five hours*. On dissection, the mucous lining of the stomach, duodenum, and a portion of the jejunum was found destroyed in patches. (Beck's Med. Jurisp., vol. ii. p. 666.)

Chemical analysis.—Bichromate of potassa is distinguished from all other salts by the deep orange-red color of its crystals. Its solution has a similar color, and possesses an acid reaction. It may be identified by the following tests: (1) *Acetate of lead* gives with it a bright-yellow precipitate (chromate of lead); (2) *nitrate of silver* yields a deep-red precipitate; (3) *sulphuretted hydrogen* gives a dingy-green precipitate. Potassa may be discovered in it by the bichloride of platinum.

CHAPTER XX.

VEGETABLE AND ANIMAL IRRITANTS.

SECTION I.

POISONING BY VEGETABLE IRRITANTS.—CROTON OIL.—ELATERIUM.—ALOES. —COLOCYNTH.—CASTOR-OIL BEANS.—COLCHICUM.—SAVIN.

THE vegetable kingdom furnishes numerous substances possessing a highly acrid, poisonous nature. Those only will be briefly noticed here that are employed in medicine, and overdoses of which have been known to occasion violent symptoms, and even death.

These irritants appear to owe their activity to the presence of either an acrid oil or a resin; in which respect they differ notably from the *neurotics* proper, in which the active principle is either an alkaloid or a neutral body. An exception to this, however, is afforded in the case of colchicum and elaterium, the former of which contains an alkaloidal principle—*colchicina*, and the latter a neutral substance—*elaterin.*

The *drastic cathartics*, as a class, are distinguished for their powerful irritant impression on the mucous membrane of the alimentary canal. In medical practice, it is customary to give them in combination with one another—small doses of each,

—by which means their individual acrimony is diminished; but if they are taken in excessive doses, and especially in a debilitated state of the system, they may produce alarming prostration, terminating in death.

The *symptoms* produced by this class of poisons are those of irritation of the alimentary canal—vomiting, purging, pain in the abdomen, cramps, tenesmus, and strangury. The patient falls into a state of collapse, attended occasionally with drowsiness and slight nervous symptoms.

The *post-mortem signs* are those indicative of inflammation of the gastro-enteric mucous membrane, in its different stages.

The most powerful drastics are croton oil, elaterium, scammony, gamboge, colchicum, and hellebore.

CROTON OIL.—This is a fixed oil, extracted by pressure from the seeds of the *Croton tiglium.* It is a prompt and powerful purgative in the dose of one or two drops. Overdoses occasion violent irritant symptoms, with collapse, resembling some of the worst cases of cholera. M. Chevallier reports two cases of poisoning by this oil. In one, a druggist swallowed, by mistake for cod-liver oil, half an ounce of croton oil. He felt a burning sensation in his throat and stomach, soon followed by vomiting and copious purging, with symptoms of collapse. He did not recover until after a fortnight. In the other case, quoted from Devergie, a man aged twenty-five swallowed by mistake two drachms and a half of the oil. The most violent purging, with collapse, took place, and the patient died in four hours. (Ann. d'Hyg., 1871, i. p. 409.) The following abstract of a case described by Dr. Greenhow (Med. Times and Gaz., Aug., 1866, p. 143) affords a good example of this form of poisoning. An old lady took by mistake an embrocation containing thirty minims of the oil. When seen two hours afterwards, she had all the appearance of a person in the cold stage of cholera. There had been profuse purging of matters exactly resembling the rice-water stools of cholera patients, together with severe cramps. The surface was cold, the features shrunken, and the skin even more blue than is usual in cases of true cholera; the pulse thready and almost imperceptible; and the respiration gasping. She was very restless, but her intellect remained unimpaired: she died ten hours after taking the poison.

We have seen several cases of the accidental swallowing of a croton oil embrocation, but where the quantity of the poison was not so great as in the instance last mentioned: after very severe symptoms of gastro-enteric irritation, recovery took place under the use of demulcents and opiates.

The poisonous properties of croton oil depend upon a peculiar fatty acid (crotonic acid), which it contains in variable quantity. When deprived of this acid, the oil is perfectly harmless.

Analysis.—Croton oil is of a light-yellow color, has a very unpleasant odor, and a hot, acrid, burning taste. It is very soluble in ether, by means of which it may be separated from other substances. When warmed with nitric acid, it turns of a dark-brown color.

ELATERIUM.—This is the product of the *Momordica elaterium*, or squirting cucumber. The juice of this fruit deposits, on standing, a sediment or fecula, which constitutes the substance in question. The English elaterium is a very active drastic purgative in the dose of one-eighth to one-fourth of a grain. It owes its activity to a neutral crystalline principle, *elaterin*, which constitutes about one-fourth of the extract. With cold sulphuric acid it gives a red-brown solution, made darker by heating. Nitric acid does not change its color.

ALOES.—COLOCYNTH.—JALAP.—SCAMMONY.—GAMBOGE.—These substances are much employed in medicine, in small doses, as active cathartics; but in large quantities they act as powerful irritants, producing violent vomiting and purging, with other symptoms of active irritation. Some of them constitute the active ingredients in several popular purgative pills.

A mixture of aloes and canella has long been known under the name of *hiera picra*, or *holy bitter:* it is used as a popular abortive, and cases are reported of serious results following its use in the pregnant female. The active principle of aloes, termed *aloin*, is soluble in water: it is distinguished by imparting to cold sulphuric acid a yellow color, which is heightened by warming it, and at a high heat changes to green. Nitric acid turns it orange.

Colocynth and jalap owe their power to neutral principles—*colocynthin* and *jalapin.* Scammony and gamboge are inspissated juices, and are classed among the gum-resins; the *resin* is the active principle.

CASTOR-OIL SEEDS.—These are derived from the *Ricinus communis* (*Palma Christi*), or castor-oil plant. By pressure, they yield the castor oil of the shops. The beans or seeds contain a powerful irritant principle, which appears to be dispelled at a certain temperature, and which, consequently, does not exist in the commercial oil when properly prepared. Two or three of these seeds, when chewed and swallowed by an adult, will generally act as an active drastic cathartic. They have occasioned death in more than one instance,—their effects being altogether disproportionate to the amount of oil contained. *Three* seeds have destroyed the life of an adult male in forty-six hours; and twenty seeds proved fatal to a young lady in five days, after violent purging and vomiting, cold skin, shrunken features, small and wiry pulse, thirst, pain in the abdomen, and serous discharges—a combination of symptoms strongly resembling those of malignant cholera. A post-mortem inspection revealed violent inflammation of the stomach and small intestines; together with abrasion of the mucous lining of the stomach and bowels. (Taylor, Prin. and Prac. of Med. Jurisp., 1873, p. 329.)

COLCHICUM (*Meadow Saffron*).—The *Colchicum autumnale*, or meadow saffron, contains a powerful alkaloidal principle, *colchicina*, which resembles veratria in many of its properties. This abounds chiefly in the bulb or corm and seeds of the plant; though the leaves and flowers are also stated to have produced poisonous effects. These effects are such as usually accompany the more active irritants, such as burning pain in the throat, great thirst, vomiting and serous purging, cramps, cold collapsed skin, small and feeble pulse, suppression of urine, and rapid exhaustion. The nervous system does not appear to be affected; the intellect remains clear, and neither convulsions nor loss of consciousness are reported among its effects.

The precise quantity necessary to occasion a fatal result is unknown: it sometimes happens that an ordinary medicinal dose will occasion alarming depression. A drachm of the wine of the fresh root has been known to produce violent irritation of the stomach and bowels. Dr. Taylor mentions a case reported to him, in which three and a half drachms of wine of colchicum, taken in divided doses, caused death on the fourth day. In another case, in which an ounce of the wine was taken, death occurred in thirty-nine hours. And in another case, a gentleman swallowed, by mistake, one ounce and a half of the wine: he was immediately seized with severe pain of the abdomen; other symptoms of irritation set in, and he died in seven hours. Mr. Fereday reports a case in which two ounces of the wine were taken: the symptoms did not come on for an hour and a half; death ensued in forty-eight hours, after violent irritant effects, but with no signs of cerebral disorder. (Med. Gaz., x. p. 161.)

A frightful accident happened in Montreal, Canada, in November, 1873, to a company of eight or ten persons. Some one of the number had procured (by theft, it is supposed) a half-gallon bottle, full of what was believed to be wine, but which was in reality *wine of colchicum*, that had been sent to some druggist's shop. Nearly all the party freely partook of it, and were made violently sick in the course of a few hours. Nausea, severe vomiting, excruciating pain of the abdomen, cramps, purging, and prostration were among the prominent symptoms. Five of the cases terminated fatally, within thirty-six hours.

The *morbid appearances* are often of a negative character; no marked evidences of inflammation are present in numerous cases; while in others there are patches of inflammation in the stomach and intestines, with softening of their mucous membrane. In one instance, the vessels of the pia mater were much injected, while there was no redness of the mucous membrane of the stomach. (Ann. d'Hyg., 1836, ii. p. 394.) The lungs have been found deeply congested.

The alkaloid *colchicina* occurs in fine, white crystals. It is soluble in water, has a feeble alkaline reaction, and a bitter, acrid taste. Its solutions give a white precipitate with tannic acid, a yellow one with chloride of platinum, and a brownish one with iodine. Its best test is *nitric acid*, which, when concentrated, produces with it a violet color, changing to blue and brown. The solubility in water distinguishes it from veratria, as also the fact of its not occasioning sneezing, like that substance. It may be procured from organic mixtures, and from the contents of the stomach and organs of the body, by the process of Stas, as described on page 110 (see also STRYCHNIA). Less than half a grain of colchicina has proved fatal. There is no known antidote. The *treatment* consists in the speedy evacuation of the poison by an emetic (mustard will answer very well), castor oil, with demulcents, opium, and stimulants.

SAVIN.—The tops of the *Juniperus sabina* abound in a yellow, volatile oil (*oil of savin*), which may be obtained by distillation. Both the powder and the oil are employed in medicine; and both possess powerfully irritant properties.

Savin is seldom, if ever, resorted to directly for its poisonous properties; but it is much used popularly for its reputed powers as an abortive: many fatal cases are recorded resulting from its employment with this view; and in the majority of instances death has resulted from the violence of the inflammation set up, without the expulsion of the fœtus. It is not believed to possess any specific powers as an abortive, the uterine contraction being due to the violent shock upon the system. In cases of poisoning by powdered savin, the latter may be recognized, after death, by microscopic examination of the portions of the leaves found in the stomach or bowels, or of the matters vomited.

The *oil* can be recovered by distilling the matters supposed to contain it, and agitating the distillate with one-third of its bulk of ether, in which the oil is completely soluble. It is recognized by its peculiar powerful, terebinthinate odor. Nitric acid in the cold slowly imparts to it a dark, red-brown color.

SECTION II.

POISONING BY BLACK, GREEN, AND WHITE HELLEBORE.

There are several species of hellebore, but the above-named alone require our attention.

The root of the black hellebore (*Helleborus niger*)—formerly named *Melampodium* — is sometimes used in medicine. It possesses drastic properties, and produces other powerful irritant effects, such as violent vomiting, pain in the abdomen, cold sweats, convulsions, insensibility, and death. In the several cases of poisoning by it, reported at different times, the most violent symptoms followed its use, resembling the collapse of malignant cholera. The post-mortem lesions were such as follow the most active inflammation. The infusion of the root and leaves, which is a popular remedy in England for worms, has proved fatal in several instances.

GREEN HELLEBORE (*Veratrum viride.—American Hellebore.—Indian Poke*).—This species of hellebore is likewise possessed of very active properties, and has occasioned violent and alarming symptoms, and, in some instances, even fatal results. The *tincture of veratrum viride* is officinal in the British and United States Pharmacopœias, and is used in medicine as a powerful depressant to the circulation.

In an instance recorded in the "American Journal of the Medical Sciences," October, 1865, p. 563, two gentlemen swallowed, by mistake, each about a tablespoonful of the fluid extract of veratrum viride. In about half an hour one of the patients was found almost speechless, retching and vomiting incessantly, bathed in profuse cold perspiration, and with a scarcely-perceptible pulse. On the administration of a teaspoonful of laudanum, the vomiting ceased, and he rapidly recovered. In the other case, where no laudanum was administered, the vomiting continued for some hours, with a total loss of speech and of locomotion for some time. A case is also mentioned where an ointment of veratrum viride applied to an ulcer on the leg, produced vomiting.

A case of poisoning by the *tincture* was mentioned to the author by Mr. George Ashmead, a druggist, of Philadelphia.

A physician, aged seventy-five, of feeble health, had accustomed himself to the daily use of the tincture in doses of eight or ten drops. On one occasion he incautiously swallowed about fifteen drops of the *fluid extract.* In about twenty minutes he went into the store and remarked that he had taken an overdose. He swallowed some tincture of ginger and laudanum, after which he vomited and retched severely. His countenance became pale and ghastly; his respiration feeble and labored; his pulse weak and fluttering, and finally could not be felt. He fell back as though dead; consciousness was not lost. Recovery gradually took place under the free use of brandy, carbonate of ammonia, and compound spirit of lavender, together with sinapisms to the feet, spine, and stomach.

According to M. Oulmont (Bul. Gén. de Thér., t. lxxiv. p. 145), veratrum viride, although resembling veratrum album in its general sedative effect, differs from the latter in its less intense action on the alimentary canal, and in leaving no post-mortem signs of inflammation in that organ.

Two active alkaloidal principles exist in this drug, named *veratroidia*—from its resemblance to veratria—and *viridia*,—the latter closely resembling, and by some believed to be identical with, the *jervina* of veratrum album. Veratroidia and viridia were believed by their discoverer, Mr. Charles Bullock, of Philadelphia (Proceed. of Am. Phar. Assoc., 1867), to be nearly identical in their relations; but they have certain distinct properties. The former is soluble in ether; the latter is not. Veratroidia, although it resembles veratria in causing sneezing, is distinguished from the latter by its higher melting-point, by its producing intense redness in contact with concentrated sulphuric acid, and by not answering to Trapp's test for veratria (see *post*).

WHITE HELLEBORE (*Veratrum album*).—This is the most poisonous of all the species of hellebore. The powdered root produces a strong local effect on the system, and causes violent sneezing. Taken internally, it produces a sense of burning heat, and constriction of the mouth and throat, great anxiety, nausea, violent vomiting and purging, pain in the bowels, trembling of the limbs, great prostration, cold sweats, very feeble pulse, followed by giddiness, dilatation of the pupils, convulsions, insensibility, and death. Some instances are recorded in which purging was absent.

In one case, related by Wibmer, twenty grains of the powdered root caused convulsions and death in three hours; and in another, a man after eating the root died in six hours. Death was preceded by vomiting of bloody mucus, and by cold sweats. Its external application to the epigastrium has occasioned vomiting, as in the case of the American hellebore (*V. viride*).

The active principle of white hellebore is the alkaloid *veratria*, which likewise exists in the *Veratrum sabadilla*, and in *sabadilla* or *cevadilla*—the seeds and fruit of *Asagræa officinalis.* The precise nature of this active principle is not yet positively settled. Two new alkaloids—*barytina* and *jervina*—were discovered in it by Simon; and these are believed by Dr. Peugnet to be identical with the veratroidia and viridia of Bullock (N. Y. Med. Record, 1872, p. 121); but this is denied by Dr. H. C. Wood (Therapeutics, 1874, p. 141).

The identity of veratria with the active alkaloid existing in veratrum viride appears to have been established by the researches of Worthington (Jour. of Phil. Col. of Phar., vol. xxix., p. 204), J. G. Richardson, Prof. S. R. Percy, and G. J. Scattergood (*ibid.*).

Veratria.—As found in the shops, this alkaloid is in the form of a nearly colorless powder. It can be crystallized, though with considerable difficulty. It has a very acrid and somewhat bitter taste, followed by a sense of dryness of the fauces. In its perfectly pure state, it is devoid of bitterness. It occasions violent irritation of the nostrils, causing excessive and prolonged sneezing.

It is insoluble in water, but more or less soluble in alcohol, ether, chloroform, benzole, and amylic alcohol. It has a slightly alkaline reaction, and forms soluble salts with the acids. When heated on porcelain, it darkens and melts into a yellow liquid, blackens and spreads into an abundant carbonaceous layer. The vapor has a disagreeable, pungent odor, and, when received on a clean dish, deposits detached crystalloids or crystals, described as rhomboidal, but among which several octahedra can be discovered. When heated on platinum-foil, the alkaloid is entirely consumed. (Guy's Foren. Med., p. 618.)

Effects.—As found in the shops, veratria varies much in strength. According to Dr. Wormley, two grains of nearly colorless commercial veratria given in solution to a cat, immediately laid it prostrate; the animal frothed at the mouth, and died in less than a minute after taking the dose. Three grains of the same preparation given to a young dog, caused immediate vomiting and purging, involuntary urination, and great prostration, followed by death in two hours after swallowing the dose. Of another sample, two grains were given to two small dogs, each, without producing any appreciable symptoms other than slight prostration.

On man, veratria is capable of producing very violent effects. Dr. Taylor (On Poisons, p. 510) mentions the case of a lady in whom one-sixteenth of a grain occasioned the most alarming symptoms, such as insensibility, cold surface, failing pulse, and collapse.

The proper *treatment* consists in speedy evacuation of the stomach, and the administration of stimulants with laudanum, or some other preparation of opium. The latter medicine is peculiarly appropriate. Tannin has also been recommended as an antidote.

The *post-mortem* lesions are not characteristic. The stomach and bowels have been found inflamed, and sometimes contracted. The lungs, liver, and heart have been seen gorged with blood.

Chemical analysis.—The most characteristic test is sulphuric acid. When the *pure* alkaloid is touched with a drop or two of the concentrated acid, it assumes a yellow color, then a reddish tint, and slowly dissolves to a pinkish solution, which, after several minutes, acquires a deep crimson-red color. These changes are brought about immediately on the application of heat. Even if the acid be very dilute, this characteristic test is brought out, by evaporating to dryness. (See SULPHURIC ACID, *ante.*)

The delicacy of this test is such that, according to Wormley, less than one ten-thousandth of a grain can readily be detected by first warming the veratrine deposit, and then adding a drop of the acid, and continuing the heat.

It has been objected to this test that other substances will give a red color with sulphuric acid,—such as *solanine*, *narceine*, *salicine*, *piperine*, etc.; but all these substances are *immediately* colored by cold sulphuric acid, whereas veratria requires the lapse of some minutes before it assumes the characteristic crimson-red tint on the application of the *cold* acid. Moreover, the colors produced with the above-named substances and sulphuric acid, under the prolonged application of heat, are different from the color produced by veratria.

Trapp's test is asserted to be even more delicate. This consists in warming the colorless solution of veratria in concentrated hydrochloric acid, when a very persistent dark-red color ensues. This test is stated to be especially useful when the veratria is impure.

Other tests mentioned in the books are *chloride of gold*, which gives a yellow, amorphous precipitate, soluble in alcohol, also on being heated; *iodine in iodide of potassium* gives a reddish-brown precipitate, soluble in alcohol; *bromine in hydrochloric acid* yields a yellow, amorphous deposit, soluble in alcohol, which, on evaporation, leaves it in the form of groups of prismatic crystals; *bichromate of potassa* throws down a yellow, amorphous precipitate, soluble in strong alcohol; tannic acid throws down a white, flocculent precipitate.

In organic mixtures.—Veratria may be separated from the contents of the stomach, and from the blood, by a modification of Stas' process, and the chloroform extract tested by sulphuric acid. Another portion dissolved in water, with a little acetic acid, may be tried with the different liquid tests above mentioned. Dr. Wormley states that by the sulphuric acid test he was enabled to recognize the presence of veratria in an ounce of the blood of a cat which had been killed in less than one minute by two grains of veratria. This shows the great rapidity with which the poison had entered the circulation. He likewise detected its presence, by means of the same test, in six fluidrachms of the blood of a dog which had died in two hours from a dose of three grains of veratria.

SECTION III.

POISONING BY CARBOLIC ACID.

This substance, in its impure state, is known as *creasote.* When pure it is in the form of delicate, white, needle-shaped crystals, which are very deliquescent; they melt at 95° F., and the oily-looking liquid boils and is entirely volatilized at 370°. It has a peculiar, powerful odor and taste, which would naturally prevent its being administered as a poison homicidally. It is procured by the fractional distillation of coal-tar, and is extensively used as a disinfectant. It has been the cause of death in several instances. In its concentrated form it acts as a powerful irritant, both externally and internally, whitening and hardening the skin and mucous membrane, and blunting the cutaneous sensibility. In one instance it is reported to have destroyed life by its external application (Brit. Med. Jour., Oct. 8, 1870).

A case of poisoning by the external use of carbolic acid is reported in the "Canada Medical Journal," July, 1870. A man aged eighty years, affected with acute eczema, which almost literally covered his whole body, was treated in the Montreal General Hospital with an ointment consisting of one part of carbolic acid to four of lard, spread upon lint: it was applied over the arms and thighs, and covered with oiled silk. In an hour and a half the man was reported to be dying. He was found in a profound coma; the pupils firmly contracted; breathing stertorous; pulse weak, rapid, and flickering; surface of the body livid; extremities cold; much mucus in the bronchial tubes; inability to swallow, and profound insensibility. By the application of powerful stimulants internally and externally, he at last recovered, after free vomiting. The peculiar odor was distinctly noticed in the vomited matters. Carbolic acid also affects the brain like a narcotic.

Symptoms.—In the concentrated state, it produces a burning sensation in the mouth, throat, œsophagus, and stomach, in the act of swallowing,—in this respect resembling the effects of the strong mineral acids and alkalies. There is violent pain in the abdomen, with vomiting of frothy mucus; cold and clammy skin; the lips, eyelids, and ears are livid; pulse small and frequent; respiration difficult, with frothing at the mouth. There is a marked odor of carbolic acid perceived in the breath, and also in the surrounding atmosphere. The pupils are contracted and insensible to light; general insensibility supervenes, which soon passes into coma, with stertorous breathing. The stools and urine, when passed, have been dark-colored,—the latter almost black.

Post-mortem lesions.—The inside of the mouth and throat is whitened, and sometimes corroded; the gullet is white, hardened, and corrugated; the lining membrane of the stomach has been found much hardened, without the usual signs of inflammation. The lungs have been found deeply congested, and the bronchi filled with a brown-red, thick mucus.

Fatal quantity.—Dr. Taylor reports the following cases. A woman died from swallowing a wineglassful of carbolic acid, —probably a weak, aqueous solution. She did not speak after taking it, and died in about half an hour. (Phar. Jour., July, 1872, p. 75.) A child died at Guy's Hospital in twelve hours after swallowing two teaspoonfuls of the ordinary brown liquid carbolic acid. In another case a tablespoonful proved fatal to a young man. In another instance an adult died in fifty minutes after swallowing one or two tablespoonfuls of the liquid acid. (See Husemann's Jahres., 1872, p. 523.)

In a case reported in the "Journal de Pharmacie et de Chimie," December, 1871, an unknown quantity of a solution of carbolic acid was swallowed by a man aged thirty-two, in mistake for wine. He was immediately seized with nausea, cold sweat, stupor, and unconsciousness. There were insensibility and contraction of the pupil; rapid, stertorous respiration, with tracheal râles; small and frequent pulse; irregular heart-beat; suppression of urine; and death from asphyxia in about nine hours.

Chemical analysis.—The strong, peculiar odor perceptible in the breath and in the matters vomited, as likewise in the body after death, will generally be sufficient to identify it. It has a slight acid reaction, and forms salts with bases. It is soluble in water and in alcohol, and gives a greasy stain to paper. It imparts a deep-violet color to perchloride of iron, and a bluish tint to ammonia and to hypochlorite of lime. Heated with the addition of cyanide of potassium it gives a red tint. The above are the best chemical tests at present known; but the peculiar *odor* is probably the most reliable criterion of its presence.

Carbolic acid has been detected in the urine both by the odor and by chemical reagents. The urine should be agitated with an excess of pure ether and allowed to stand; the ethereal layer is then to be removed by a pipette and evaporated in a glass vessel. A minute oily residue is left, having the physical characters of carbolic acid. This, when dissolved in water, will yield the above-mentioned chemical reactions.

As an *antidote* in carbolic acid poisoning, Dr. T. Husemann recommends a saturated solution of saccharate of lime. (Neues Jahres. für Pharm., Sept., 1871.)

The root of the Yellow Jessamine (*Gelsemium sempervirens*), growing in the Southern States, has been found by Dr. Wormley to contain a very powerful, poisonous alkaloid, *gelseminia:* one-eighth of a grain given hypodermically killed a rabbit in an hour and a half; there were great prostration, inability to move, gasping respiration, dilated pupils, but no convulsions. He also discovered another organic principle —*gelseminic acid.* This is crystalline, and when treated in the solid state with a drop of concentrated nitric acid it becomes yellow or reddish, according to the quantity employed. When an excess of ammonia is added, it acquires a blood-red color. The solution in potassa is fluorescent, presenting a deep-blue color on the surface. Gelseminic acid was thus detected in the contents of the stomach some months after death. (Amer. Jour. of Pharmacy, Jan., 1870.)

Yellow jessamine has been considerably employed in medicine, particularly in the Southern States. Several cases are recorded of fatal results from its administration.

Prof. Wormley has reported a fatal case of a young, healthy married woman, several weeks advanced in pregnancy, who took three teaspoonfuls of the extract. Two hours afterwards she complained of pain in the stomach, nausea, and dimness of vision. These symptoms were soon succeeded by great restlessness, ineffectual efforts to vomit, and free general perspiration. In about five hours, the pulse was feeble, irregular, and sometimes intermittent; there was great prostration, with irregular breathing and slow respiration; the skin was dry, extremities cold, pupils expanded and insensible to light, the eyes fixed, and inability to raise the lids. Sinking gradually took place without convulsions, and death occurred in about seven and a half hours after the poison had been taken. Judging by comparison with other samples experimented upon, Dr. Wormley estimated that the quantity of the alkaloid *gelseminia* swallowed could not have exceeded one-sixth of a grain. This would seem to indicate that this alkaloid is one of the most potent poisons known.

The *post-mortem appearances* in this case presented nothing peculiar. The membranes and substances of the brain and medulla oblongata were normal; the lungs natural in appearance, with the superficial veins congested; the heart normal in size, its superficial veins injected, and its cavities greatly distended with dark, grumous blood, inside of which was found a well-defined membrane, identical in appearance with that found in diphtheria and pseudo-membranous croup; the stomach, intestines, peritoneum, liver, all healthy; the left kidney congested. Dr. Wormley succeeded in detecting both the alkaloid and the peculiar acid (*gelseminic*) in the contents of the stomach (Am. Jour. Med. Sci., April, 1870, p. 531); although these had undergone considerable decomposition, and the examination was not made for several months after death.

SECTION IV.

POISONOUS MUSHROOMS (FUNGI).

Among the numerous species of *Fungi* many are edible, and many are poisonous. In this country and Great Britain only a very few species are regarded as wholesome, viz., *Agaricus campestris*, or common mushroom; *Tuber cibarium*, or common truffle; and *Morchella esculenta*, or morelle. In France and Germany, and especially in Russia, a large number of other species are considered wholesome, and, in fact, constitute an important part of the common food of the people.

The following facts may be regarded as established concerning these vegetable products. Certain fungi usually considered noxious become safe after being dried: this is the case, according to Foderé, with the *Agaricus piperatus* (Méd. Lég., iv. 61). Boiling in water has a still more decided effect. According to Dr. Pouchet (Jour. de Chim. Méd., 1839, p. 322; quoted in Christison on Poisons), the poisonous principle of two of the most deadly fungi, *Amanita muscaria* and *A. venenata*, may be completely removed by boiling in water. A quart of water in which five plants had been boiled for fifteen minutes killed a dog in eight hours, and again another in a day; but the boiled fungi themselves had no effect on two other dogs, and a third which was fed on boiled *amanitas* for two months actually fattened on this food. From the above facts, it would appear that the poisonous principle of these fungi is of a volatile nature, and capable of being dissipated by heat. Climate and cultivation appear also to influence their deleterious properties: thus, according to the same authority, the *Agaricus piperatus*, the *A. acris*, and the *A. necator*, which are considered to be poisonous in Britain and France, are freely eaten in Russia; and the *Amanita muscaria*, which is regarded even in Russia as violently poisonous, is used in Kamschatka for preparing an intoxicating beverage.

So, on the other hand, our common edible fungi appear at times to acquire noxious properties in very moist seasons, and towards the close of summer and autumn.

It must not be forgotten that certain persons cannot eat the most harmless mushrooms without suffering severely from irritation of the stomach and bowels, together with narcotic symptoms. This is, however, dependent on idiosyncrasy: the same thing occurs in reference to other kinds of diet, more particularly certain varieties of fish.

It is impossible to determine, at present, what is the precise character of the poisonous principle of the noxious fungi, or whether this principle is identical in them all. M. Braconnot ascertained that some of them contain a saccharine matter, others an acrid resin, and others again a volatile acrid principle: they all contain a spongy substance, to which the name *fungin* has been given. Later, M. Letellier discovered in some of the fungi two poisonous principles: one, a very volatile acrid matter, easily removed by boiling and drying, or by weak acids, alkalies, and alcohol; the other principle more fixed, as it resists heat and the above solvents, but is soluble in water, and is capable of forming crystallizable salts with acids. To this latter substance he attributes the narcotic effects of the fungi. (Arch. Gén. de Méd., xi. p. 94.)

Symptoms.—The effects of poisonous mushrooms on man are those of the narcotico-irritants; that is, they occasion violent vomiting, purging, pain in the abdomen, thirst, anxiety, and cold sweats, together with giddiness, dimness of vision, trembling, staggering as if from intoxication, delirium, disposition to rave and wander about, illusions, stupor, coma, dilatation of the pupils, and convulsions, especially in fatal cases.

It is somewhat singular that the very same fungi have acted on some members of a family as irritants merely, and on others as narcotics. Generally speaking, when the narcotic symptoms are the more prominent, they come on very soon after partaking of the noxious variety,—within an hour or two; but when the irritant effects are the more evident, often these do not appear for many hours after eating,—sometimes later than twenty-four hours; and in these cases narcotism is apt to follow the irritant operation. Orfila (Toxicol., ii. p. 433) relates the following interesting case of poisoning of a family of six persons by the *Amanita citrina.* The wife, the servant, and one of the children had vomiting, followed by deep stupor; but they recovered. The husband had violent cholera; he recovered also. The two other children became profoundly lethargic and comatose; emetics had no effect, and death soon ensued. The individuals who recovered were not completely well till three weeks after the fatal repast. Dr. Taylor relates a similar result from the same poisonous fungus (Med. Jurisp., Am. ed., 1873, p. 231). The above cases show the tendency of the poisonous fungi to cause in one person pure irritation, and in another pure narcotism.

The *morbid appearances* that have been observed in persons thus poisoned are imperfectly described. The body is generally livid; the blood fluid; cadaveric rigidity is absent; there are numerous ecchymoses in the serous membranes and the parenchymatous organs; decomposition of the tissues; signs of violent, and even gangrenous, inflammation of the stomach, and congestion of the cerebral vessels.

The only medico-legal interest connected with this subject is in the fact that the symptoms occasioned by eating poisonous fungi might be attributed to some other poison, homicidally administered. A microscopic examination of the contents of the stomach and bowels will usually reveal the botanical character of the fragments of the fungi, if the poisoning has been due to them. Sir R. Christison (On Poisons, p. 929) mentions a remarkable case, which shows how easily criminal poisoning might be accomplished by mingling arsenic, for example, with some of the noxious fungi, eaten at a meal. A servant-girl poisoned her mistress by mixing arsenic with a dish of mushrooms. She died in twenty-four hours, after suffering severely from vomiting and colicky pains. Dissection revealed inflammation of the stomach, with gangrenous spots, clots of blood, and redness of the intestines. The death was, however, ascribed to the unwholesome mushrooms; and the real cause was not discovered till thirteen years afterwards, when the culprit confessed the crime.

(For an excellent and full description of the Medicinal and Toxicological Properties of the Cryptogamic Plants of the United States, consult Dr. F. Peyre Porcher's Essay, in Trans. of the American Association, vol. vii. See also Orfila and Christison, on the subject of Poisonous Fungi.)

CHAPTER XXI.

ANIMAL IRRITANTS.

CANTHARIDES.—POISONOUS ANIMAL FOOD.—SAUSAGE-POISON.—TRICHINIASIS. —CHEESE-POISON.—POISONOUS FISH.—MUSSELS.—PUTRESCENT FOOD.—POISONED FLESH.

SECTION I.

POISONING BY CANTHARIDES.

THE *Cantharis vesicatoria*, or Spanish fly, is much used in medicine, externally as a vesicant, and also sometimes internally. In overdoses it is capable of producing very violent, and even fatal, effects, by its powerful irritant action on the gastro-intestinal mucous membrane, and on the genito-urinary organs; and in fatal cases, causing decided cerebral symptoms. The fly owes its active properties to a peculiar crystalline principle termed *cantharidin*, which exists in the proportion of about one grain to half an ounce of the powder. (Guy's Forensic Med., p. 631.)

The Spanish fly is distinguished by the shining, golden-green color of the head, legs, and wing-cases. It is kept in the shops in the form of powder, tincture, liniment, and plaster. The powder and plaster are readily identified by the numerous shining, golden-green particles which they contain. Prof. Guy (*loc. cit.*) recommends a very simple mode of distinguishing the powder, namely, by heat. A very minute portion—even the one-thousandth of a grain—is placed on a flat porcelain slab (a small crucible-lid reversed answers well), within a ring of glass, and a glass disk is laid over it. On raising the temperature to about 212° F., a white sublimate appears on the glass disk; and when this is examined by the microscope, it is found to consist of crystals of *cantharidin.* If the sublimate be amorphous, or not distinctly crystalline, it should be dissolved in a few drops of ether, which, on evaporation, will deposit it in the crystalline form.

Symptoms.—When taken in the dose of two or three drachms of the *powder*, or of one or two ounces of the *tincture*, cantharides occasion a burning sensation in the mouth and throat, great difficulty of swallowing, with a sense of constriction. This sensation speedily extends to the gullet and stomach, and is succeeded by violent pain in the abdomen, increased by pressure. There are nausea and vomiting of bloody mucus and shreds of membrane, along with great thirst and dryness of the fauces. Very soon the characteristic impression on the genito-urinary organs displays itself; there is a heavy, dull pain in the loins, an urgent and incessant desire to urinate, which is attended with great pain and the voiding of merely a few drops of bloody urine, accompanied with tenesmus. Priapism frequently occurs in males, with occasional satyriasis and seminal emissions; and swelling and heat of the labia in women, together with abortion, in the pregnant condition.

It has been stated that the female genital organs are frequently attacked with gangrene, even in cases where no sexual excitement has been manifested. (Wharton and Stillé, Med. Jurisp., 1873, ii. p. 474.)

Purging generally supervenes, the matters discharged being mixed with blood and mucus, and accompanied with tenesmus. A careful inspection of the discharges from the stomach and bowels will generally reveal the shining green particles already alluded to, if the poison has been swallowed in the form of powder. Sometimes profuse salivation occurs; and in fatal cases death is preceded by faintness, giddiness, and convulsions. Cantharides have the popular reputation of possessing *aphrodisiac* properties; and they have been frequently administered with the view of exciting the sexual passions,—and sometimes with serious, and even fatal, consequences.

When the tincture has been taken, the symptoms come on more rapidly; and the burning and constriction of the mouth and throat, together with the difficulty of swallowing, are more strongly marked.

All the above symptoms, even to the fatal result, have been produced by the external application of this poison. The painful effect on the genito-urinary organs, termed *strangury*, occasioned in many persons by the application of an ordinary fly-blister, is familiar to every physician. Dr. Taylor (On Poisons, p. 513) mentions the case of a young girl who was killed by the external application of a blistering ointment, that was rubbed over her whole body in mistake for sulphur ointment, which had been prescribed for the itch. Although the ointment was washed off, the cuticle came off with it, and the girl died in five days with the symptoms above described. Guibourt has reported a case of a young man suffering from pleurisy, who had a large blister applied to his side: the result was violent irritation of the urinary passages, followed by collapse and death. (L'Abeille Méd., xv. 153.)

Fatal quantity.—The medicinal dose of cantharides is one to two grains of the powder, and ten to thirty drops of the tincture, gradually increased until slight symptoms of strangury are produced. But, as the powdered flies speedily deteriorate, it frequently happens that all the preparations of cantharides, as found in the shops, are nearly, if not quite, destitute of active properties,—a circumstance that will account for the large quantities that have been taken without producing serious results. The smallest quantity of the powder that has destroyed life is recorded by Orfila, in the case of a young woman, who aborted and died in four days after taking *twenty-four grains* in two doses. In this instance, however, the death may have been indirectly due to the abortion. *An ounce* of the tincture occasioned death in fourteen days, in a boy aged seventeen years.

Treatment.—There is no antidote to this poison. It should be removed as speedily as possible from the system by the free use of emetics and cathartics. Opium, in the form of enema or suppository, is especially advantageous for relieving the painful strangury; and likewise the free use of demulcent drinks. Leeches, and other applications to the abdomen, may be required.

Post-mortem appearances.—The whole alimentary canal has been found in a state of violent inflammation. The lining membrane of the mouth and throat has been completely destroyed. In one case, in which life was prolonged for fourteen days, the mucous membrane of the stomach was not inflamed, but it was pulpy, and easily detached. Generally, the ureters, kidneys, and bladder are more or less inflamed. The brain has been found congested. The most satisfactory post-mortem evidence of the nature of the poison is the presence of the green, metallic-looking points scattered over the gastro-intestinal mucous membrane. These may often be recognized by the naked eye, and always by the aid of a good lens. The suspected liquids, after being mixed with alcohol, may be suffered to evaporate on a plate of glass, which will enable the observer to identify the shining-colored particles, when dry. Or, the stomach and intestines may be inflated and dried, after which, on cutting them open and examining them upon a flat surface, the shining points may be observed closely adherent to the mucous membrane.

According to Orfila, the powder is not affected by putrefaction, it having been recognized nine months after death. If, however, the tincture has been taken, then, of course, this physical examination would be impracticable. In this case it would be necessary to endeavor to procure the evidence of *cantharidin*, by the means to be presently described.

Chemical analysis.—The *powder* is to be identified in the manner above pointed out. If this cannot be done, the suspected solids and liquids should be dried and digested in successive portions of ether until exhausted. This will dissolve out the *cantharidin.* The ethereal solution is to be evaporated to an extract, and some of the latter, spread upon oiled silk, should be applied on the lips, or on a thin portion of the skin of the arm, when the resulting vesication would denote the presence of cantharidin. Chloroform may be used in the place of ether.

Cantharidin occurs in colorless crystalline plates, of various forms and thickness. It may be identified by the action of heat (212° F.), causing it to sublime in crystals, as before described (*ante*, p. 349). Again, the negative action of both sulphuric and nitric acids upon it serves to distinguish it from all the poisonous alkaloids or neutral principles. Further, its vesicant property will serve to identify it. The one-hundredth of a grain, dissolved in ether, is said to possess vesicating powers.

SECTION II.

POISONOUS ANIMAL FOOD.—SAUSAGE-POISON.—TRICHINIASIS.—CHEESE-POISON.

It occasionally happens that certain kinds of animal food will produce violent symptoms in persons partaking of it, resembling those of an irritant poison. Sometimes this effect can be explained by an idiosyncrasy of the patient; but where several persons partaking of the same food are simultaneously affected with the same symptoms, such an explanation will not suffice, and the cause must be referred to some noxious agent, either introduced from without, or else inherent in the food itself. Such cases may readily come within the scope of a medico-legal inquiry, as the symptoms might very naturally be referred to the effect of an irritant poison administered with criminal intent.

The articles of food that have been the most frequent sources of irritant poisoning are sausages, cheese, diseased pork, certain shell-fish, especially mussels, poisoned flesh, and diseased or putrefying flesh.

SAUSAGE-POISON.—The precise nature of this animal poison has never been determined. It is of an acrid, narcotic nature, and produces very alarming symptoms, which may result fatally. They are usually slow in appearing, several days elapsing before they are developed. It is chiefly in Germany, where the sausages, after being cured and dried, are generally eaten uncooked, that the most formidable cases occur. According to some authorities, the poisonous principle is the result of a partial decomposition or putrefaction, determining the production of a peculiar fatty acid, named by Buchner *botulinic acid.* This is a non-volatile product, soluble in alcohol, insoluble in water, having a peculiar nauseous odor and disagreeable, oleaginous taste, followed by an extraordinary dryness of the throat for several hours. It proved fatally poisonous to dogs in the course of a few days. Physicians and physiologists of the present day are disposed to attribute the formidable effects of sausage-poison to a certain species of entozoon named *trichina spiralis*, which especially infests the muscles of the pig; and which, when the pork is eaten by man, unless it has been thoroughly cooked by being exposed for a long time to a temperature above 212° F., very soon penetrates the muscular coat of the intestines, and thence spreads through the muscles generally. It has been ascertained by repeated researches that the trichina is a viviparous parasite, which passes the greater part of its existence in the chrysalis state in the muscles of the pig. They appear as small, ovoid bodies, or capsules, resembling white specks, to the naked eye, but distinctly perceptible by means of a magnifier. They are often so numerous as to give the flesh a speckled appearance. There can be no doubt that much of what is termed *measly pork* is really of a trichinous nature. According to Dr. Keller, as many as three hundred thousand have been counted in half a pound of raw meat. When this flesh is eaten as food, the parasite finds in the human stomach a proper medium for its full development into a worm. The period of incubation in the stomach and bowels of man or of warm-blooded animals, is from six to eight days; and during this time it there thrives and propagates to an almost incredible extent. Dr. Keller states that in three or four days a single female produces one hundred or more young ones, which begin in the sixth day to leave the parent animal; and he estimates that in a few days after eating half a pound of infected meat, the stomach and intestines may contain thirty millions of these worms.

When once introduced into the alimentary canal, these worms leave their capsules, produce young which migrate through the walls of the intestines into the various muscles of the body, where they become encysted in new capsules formed at the expense and by the destruction of the muscular tissue. The sudden liberation of a multitude of these parasites produces the characteristic irritation of the bowels, and the subsequent loss of muscular power, so generally witnessed in the disease.

The *symptoms* of *trichiniasis* usually manifest themselves within a few days after the ingestion of the diseased meat. There is first loss of appetite, pain in the bowels, purging, sickness, prostration, swelling of the eyelids and joints, profuse, clammy perspiration, and fever of a typhoid character. Death results either from paralysis, or from peritonitis and irritative fever. No mode of medical treatment has availed to arrest the disease, when it is once established in the system.

It might readily happen that the symptoms above described might be attributed to slow poisoning by one of the mineral irritants: hence the importance of a proper understanding of this subject, on medico-legal grounds. A careful microscopic examination of the suspected food, and, in case of death, of a piece of the muscle, will reveal the true parasitic cause of the disorder. (For a full account of *trichiniasis*, see paper by Dr. Keller, of Darmstadt, also Dr. Lücke's paper, in Casper's Vierteljahr., 1864, No. 1, p. 103, and No. 2, p. 269; also, Dr. Dalton's paper, in N. Y. Med. Record, April 1, 1869; also, Chicago Med. Jour., Aug., 1866; and Canada Med. Jour., 1870–1.)

CHEESE-POISON.—The symptoms which occasionally result from eating cheese strongly resemble those of an irritant poison. What is the precise nature of the noxious material in the cheese, has never been positively determined. In some instances, where a number of persons have suffered from severe cholera morbus after partaking of cheese, it has been impossible to discover any injurious substance, either of an inorganic or organic nature, on the most careful analysis. In other instances, the morbific cause has been traced to a chemical change arising from an imperfect fermentation of the curd, whereby certain new products of an irritant quality are generated. These are believed by Hünefeld and Sertürner to be analogous to, if not identical with, caseic and sebacic acids. In the process of fermentation in cheese-making, there is a gradual conversion of the casein into the caseate of ammonia, which, in some cheese, is always united with excess of alkali. But if the fermentation has been too much hastened, or allowed to go too far, a considerable excess of caseic acid is formed, as well as some sebacic acid. According to Braconnot, the caseic acid of Proust is only a modification of acetic acid and an acrid oil. It is certain that such an oil can be extracted from the poisonous cheese by means of ether.

According to Hünefeld, the injurious cheese is yellowish-red, soft, and tough, with harder and darker lumps interspersed; it has a disagreeable taste, reddens litmus, and becomes flesh-red, instead of lemon-yellow, under the action of nitric acid. The instances of cheese-poisoning are much more common in Germany than in either England or the United States.

The *symptoms*, as they appear in man, are very similar to those caused by sausage-poisoning: they usually come on within a few hours after the cheese is eaten. These have sometimes been attributed, erroneously, to the accidental impregnation of the milk by copper; and sometimes they have been traced to noxious vegetables eaten by the cows. (See Christison on Poisons, pp. 641–2.)

SECTION III.

POISONOUS FISH.—MUSSELS.

Many kinds of fish prove poisonous, *i.e.* they excite severe gastro-intestinal symptoms, resembling cholera morbus, in certain persons. This may depend either on idiosyncrasy in the patient, or on some peculiar, undiscovered organic change that has taken place in the food itself. Sir R. Christison considers "the subject of fish-poisoning to be one of the most singular in the whole range of toxicology; and none is at present veiled in so great obscurity." (On Poisons, p. 618.)

Among the shell-fish which are commonly nutritive but which sometimes acquire poisonous properties, the *common mussel* is the most conspicuous. In many parts of Europe, this shell-fish is used considerably as a common article of food; but on various occasions, without the possibility of being able to ascribe it to any definite or rational cause, it has occasioned the most violent and alarming symptoms, which have not unfrequently resulted in death.

The effects produced by eating poisonous mussels are not uniform. Sometimes they are those of a simple irritant, occasioning nausea, vomiting, pain in the stomach, purging, cramps, and a small, quick pulse. The fatal cases disclosed, on post-mortem examination, evident signs of inflammation.

In other instances the gastro-enteric symptoms have been slight, while the constitutional disturbance has been more marked, the most conspicuous symptoms being a peculiar eruption like *nettle-rash*, and a violent asthma. Other cases, again, have been attended with dyspnœa, lividity of the face, delirium, insensibility and convulsive movements of the extremities, and coma. As a rule, the symptoms do not come on until about twenty-four hours; but in some exceptional instances they have made their appearance within a few minutes. Most of the cases appear to have recovered under the use of emetics. In most of the fatal cases, no appearance was discovered after death to account for the result.

The following cases are referred to by Dr. Taylor (Med. Jurisp., 1873, vol. i. p. 339). A woman picked up some mussels which she found at the bottom of the basin of a ship-canal. She distributed them among her neighbors, and during the night twenty-one persons who had eaten them were attacked with symptoms of poisoning. Three children died, and six persons were placed in imminent peril; the rest were soon out of danger. In October, 1862, an accident occurred at Liverpool, in which a woman died in about four hours after eating mussels that had been taken from a ship in the docks. Severe pain and vomiting were among the symptoms, which generally resembled those of arsenic-poisoning. Several other persons were made seriously ill, but recovered.

As to the *cause* of the noxious properties in mussels, various opinions and speculations have been formed. It has been vulgarly ascribed to the presence of *copper*, which has been supposed to have been received from the copper sheathing on the bottoms of ships. This idea is, however, untenable, since chemical analysis has failed to detect the presence of this metal in the majority of the cases. Another theory refers the cause to putrefaction; but this cannot be sustained, inasmuch as in nearly all the cases reported the mussels were perfectly fresh. Still another theory ascribes the poisonous principle to some disease of the fish; though no one has yet been able to determine what this disease is. Neither does the locality where the mussel has been found throw any light upon the subject, since some have been attached to wooden logs, some to rocks, and some to the stones of a dock. Nevertheless, according to Dr. Combe's description of the poisoning at Leith, every person who ate of the mussels taken from a particular spot were more or less severely affected; and in this last instance certain animals suffered as much as man, a cat and a dog having been killed by eating the mussels. From what has been said, it is evident that the poisonous effects must be due to some peculiar *animal* principle, either generated, or developed under unknown conditions. Other shell-fish besides mussels have occasionally given rise to similar symptoms.

SECTION IV.

POISONING BY PUTRESCENT FOOD.—UNSOUND MEAT.

The effects resulting from eating meat that has undergone putrefaction must not be confounded with those caused by a diseased condition of the animal. The meat of the most healthy animal after it has become putrescent is poisonous to man. It not only produces, in common with the flesh of diseased animals, symptoms of irritant poisoning, but also, in addition, those of a typhoid character, or *septicæmia*, indicating a true blood-poisoning. The general *symptoms* arising from partaking of such food strongly resemble those of some of the irritant mineral poisons, and might readily give rise to the suspicion of such poisoning. There are vomiting and purging, giddiness, heat of the throat, general numbness, with redness of the eyes. The early vomiting that is excited usually seems to expel the noxious substance from the system. In the matters vomited, the presence of sulphuretted hydrogen can readily be detected. The tendency of putrefaction to impart deleterious qualities to animal matters originally wholesome, has long been known. To those not accustomed to the use of tainted meat, the very commencement of decay is enough to render it disgusting to the taste, and highly irritating to the stomach and bowels. The game that has been kept long enough to delight the taste of the epicure, has produced a severe cholera in persons not accustomed to its use.

Putrid animal matter, when injected into the blood, proves quickly fatal, after causing typhoid symptoms. It is well known that the putrid animal matter of the dissecting-room entering the blood through an abrasion of the skin, causes the most alarming symptoms, which often terminate fatally. There is extensive local inflammation of the veins and absorbents, together with diffusive cellular inflammation, and great constitutional fever of a low character.

Even the emanations of decaying animal matter have proved fatal to dogs that were made constantly to breathe the exhalations; and doubtless similar effects would be produced on man, if exposed to the like noxious influences. It is highly probable that certain low fevers, very similar in their symptoms and general history to typhoid fever, may be excited by constantly breathing an atmosphere tainted by the impure emanations arising from decaying animal matters, as, for example, from privies. Yet, on the other hand, as if to refute the idea of the emanations from putrefying animal matter being unwholesome, we are pointed to the famous establishment at Montfaucon, close by Paris, where the contents of the city privies are collected, along with thousands of bodies of horses, dogs, and cats; the whole decomposing together, and giving rise to a stench perfectly insupportable to the uninitiated; and yet the workmen and their families appear actually to thrive upon these exhalations, since they are stout, healthy, and long-lived.

Sir R. Christison cites an instructive instance of the poisonous effects resulting from eating decayed flesh. Four adults and ten children partook of a stew made from meat taken from a dead calf that had been found on the sea-shore. After the lapse of three hours, they were all seized with pain in the stomach, efforts to vomit, purging, and lividity of the face, succeeded by a soporose condition, like the stupor caused by opium, except that when roused the patient had a peculiar wild expression. One person died comatose in six hours; the rest eventually recovered, after free purging and vomiting; but they required the most powerful stimulants to counteract the exhaustion and collapse which followed the stupor (On Poisons, p. 647). The conjecture is thrown out that, in consequence of the animal having long lain in the water, an adipocerous degeneration had commenced, and that in the course of the putrefaction some poisonous principle, like that of cheese or of German sausage, had been developed (*ibid.*).

M. Lassaigne has examined chemically the putrid matter formed by keeping flesh in close vessels, and has found it to consist of carbonate of ammonia, much caseate of ammonia, and a fetid volatile oil,—the last of which is probably the poisonous ingredient (*ibid.*).

Poisoned Meat.—The cases above considered are altogether different from those in which the flesh of an animal which has been previously poisoned by arsenic, strychnia, or some other substance, has been the cause of the poisoning in man. Birds may thus become poisoned by feeding on grain that has been steeped in a solution of arsenic previous to sowing. In some of these cases, where the game has been kept for any length of time, it may be a question whether the poisoning is to be ascribed to the noxious substance fed upon, that has impregnated the flesh of the animal, the latter being in a perfectly sound condition, or to the results of a putrefactive change. It is quite certain that the body of an animal may become the vehicle or medium for conveying a poison to another, which has proved wholly innocuous to itself (see *ante*, p. 86). Frequent cases of poisoning have occurred in this country from eating the flesh of the common pheasant (*Tetrao umbellus*) during the winter season. This is usually ascribed to the birds having eaten the leaves and buds of the laurel (*Kalmia*): these have been found in the crops of the birds. But, as the symptoms are almost identical with those caused by putrescent food, it would not, probably, be entirely safe to insist upon the former reason.

CHAPTER XXII.

CLASS II.

NEUROTIC POISONS.

THIS Class embraces the second general division of poisons, viz., those whose effects are displayed chiefly on the great nerve-centres. It includes the *narcotics* proper, or such as influence the brain primarily; the *spinants*, or those that affect the spinal cord; and the *cerebro-spinants*, or those whose impression is directed to both these nervous centres. The most prominent symptoms are drowsiness, headache, giddiness, stupor, delirium, convulsions of various kinds, and paralysis. These all point unmistakably to the brain and spinal marrow as the organs affected. This class of poisons produces little, if any, local irritant impression on the stomach and bowels, in which respect they differ totally from those already considered under the first class—the Irritants. The morbid appearances are by no means well marked, or characteristic. A fullness of the vessels of the brain and its membranes, with, rarely, effusion of serum, and still more rarely of blood, is, for the most part, all that can be distinguished; and as these very lesions are common as the results of various cerebral diseases, it follows that it is impossible to diagnosticate the case as one of neurotic poisoning by these lesions *exclusively*. This Class, for the sake of convenience of reference, will be considered under several Orders and Subdivisions.

ORDER I.—CEREBRAL NEUROTICS.

SECTION I.

NARCOTICS.

POISONING BY OPIUM.—MORPHIA.

Opium and its preparations constitute the most frequent of all the causes of poisoning, both in this country and Great Britain. In the latter, according to Dr. Taylor, three-fourths of all the deaths by opium take place among children under five years of age. This, however, forms but a small proportion of the total number of cases, since there is no other kind of poisoning wherein recoveries are so frequent.

Opium is a vegetable extract, the inspissated juice of the unripe capsules of the *Papaver somniferum*, or white poppy. Its odor is strong and peculiar; its taste, bitter and narcotic. It imparts its virtues to water and alcohol. It has a very complex composition, which, moreover, differs somewhat in the several commercial varieties. Along with gum, resin, coloring-matter, and inorganic substances, it contains numerous crystallizable organic bodies, the most important of which are *morphia*, *narcotina*, *codeia*, *narceine*, *meconin* or *opianyl*, and a peculiar acid, the *meconic*. Of these, the most important and interesting in a medico-legal point of view are morphia and meconic acid. In fact, in a medico-legal inquiry in a case of opium-poisoning, it is to the detection and identification of these two substances that the chemical analysis is directed.

As the poisonous properties of opium depend chiefly on the contained *morphia*, it is well to remember that the amount of this alkaloid varies considerably in different specimens of the drug,—from two per cent. (in Bengal opium) to six or eight per cent. (average in ordinary Smyrna opium). In the latter variety the quantity varies from three per cent. to over thirteen per cent. This variation in the proportion of the active principle will satisfactorily account for the discrepancy resulting in the effects produced by similar doses of the preparations of opium. This is especially noticeable in *Laudanum*, which is the ordinary tincture of opium. When this preparation is made according to the officinal formula, each fluidrachm should represent about five grains of opium,—which is equivalent to one grain for twenty-five drops. But the strength of this tincture is, of course, much influenced both by the quality of the opium and by the strength of the spirit used, and also by the period of maceration; consequently, the laudanum of the shops varies greatly in its strength.

The other preparations of opium in common use as medicines are the *Camphorated Tincture*, or *Paregoric Elixir*, which contains two grains of opium to the fluidounce; *Acetum Opii*,—

made to take the place of the old *Black Drop*, which is about double the strength of laudanum; *Wine of Opium* (*Sydenham's Laudanum*), which is about the strength of laudanum; *Battley's Sedative Solution*, which is of unequal strength, but is more active than laudanum; *Dover's Powder*, which contains one grain of opium in every ten; *Aromatic Powder*, which contains one grain of opium in forty; the *Compound Kino Powder*, which contains one grain in twenty; and some others of less importance. The *Extract* may be regarded as a very pure form of opium; it contains a larger proportion of morphia: three grains are usually considered as equivalent to five of the crude drug. The medicinal dose of opium is from one to three grains.

Symptoms.—These vary considerably, according to the size of the dose. If opium be taken in a full but not poisonous dose, there is at first a period of general excitement of the whole system, as evidenced by an increase of the force and frequency of the circulation, a warm skin, flushed face, and brilliancy of the eye; also increased activity of the brain, as denoted by a more vivid imagination and greater loquacity. This soon gives place to a period of calm repose; which in its turn is succeeded by the soporific stage, the patient falling into a profound sleep, which lasts for several hours, during which there is general relaxation of the system. In proportion as the amount of opium is increased, the first period of excitement or exhilaration is diminished; so that when the dose is sufficiently large to produce death, this first stage may not be perceived at all, but the more characteristic soporific effects will manifest themselves very early. In such a case, there will be dizziness, drowsiness, rapidly passing into deep sleep or stupor, from which there is difficulty in arousing the patient: this stupor gradually passes into complete insensibility. The profound stupor not preceded by delirium is characteristic of opium-poisoning. When under its full influence, the patient lies in a deep lethargy; the eyes are closed; the pupils most generally contracted, and insensible to light; the pulse full and slow; respiration slow and stertorous; the skin warm; and the face rather flushed. As the case advances, the countenance becomes pale and ghastly;

the lips livid; the skin cold and clammy; the respiration very slow,—sometimes, as we have noticed, amounting to only five or six in a minute; the muscles are relaxed; the lower jaw drops; the pulse becomes feeble, and scarcely perceptible; the sphincters relax, and sometimes convulsions occur just before death; these, however, are more common in children than in adults. Sometimes there is vomiting, and even purging; and if free vomiting takes place early, there is a good hope of recovery. This symptom is generally observed when a very large dose has been taken. In some cases during the comatose state the skin, though cool, is bathed in a profuse perspiration. If recovery occurs, there are nausea and vomiting, with more or less headache, together with a general itching of the skin.

Opium has the property of diminishing all the secretions, with the exception of that of the skin, which it tends to increase: hence the copious perspiration generally observed in cases of poisoning by this drug.

There are occasional variations in the symptoms which deserve attention. The pupils are sometimes *dilated* instead of being contracted: this is more apt to occur in the advanced stage of the case. Occasionally one pupil may be contracted and the other dilated. Dr. Taylor alludes to a case of this kind (On Poisons, p. 520). The reflex function is active and easily excited, although the general insensibility is complete. The pulse is sometimes quite natural; but towards the fatal termination it becomes small, frequent, and irregular; severe tetanic spasms, with difficulty of swallowing, and partial opisthotonos—symptoms strongly resembling those of strychnia—have been noticed in a case of poisoning by acetate of morphia (Med. Times and Gaz., March 7, 1857). In some instances there has been a remarkable absence of all narcotic symptoms, with sudden death; and in others, a long postponement of the symptoms, together with a partial recovery and a fatal relapse.

The contracted state of the pupils, which is generally regarded as a diagnostic sign of opium-poisoning, may likewise unquestionably result from disease. Mr. Wilks alludes to the fact that in apoplexy which is seated in the pons

Varolii the pupils are also contracted. He describes two cases of this form of apoplexy which were mistaken for poisoning by opium in consequence of this condition of the pupils (Med. Times and Gaz., 1863, i. p. 214).

First appearance of symptoms.—This depends, in some degree, on the condition of the stomach at the time, whether full or empty, and also on the amount and the form of the narcotic as swallowed, whether liquid or solid. When in large quantity, and in the fluid state, the poison may begin to act in a few minutes, and coma may be fully established in half an hour. As a general rule, the symptoms usually commence in an adult *within an hour* after swallowing the poison; but there is considerable variety in this respect. Thus, in a case quoted by Dr. Taylor, the patient was found totally insensible in *fifteen minutes.* We have more than once witnessed profound sleep produced within five minutes, in a patient suffering from violent pain, from the hypodermic injection of a quarter of a grain of morphia. Christison refers to several cases in which the symptoms were manifested within the above time; and to one in which the sopor was fairly established in fifteen minutes after swallowing two drachms of solid opium; although in another remarkable case, where *eight ounces* of solid opium were taken, an interval of an hour elapsed before any symptoms were observed (On Poisons, p. 706). On the other hand, the same author alludes to an extraordinary case communicated to him, of a man swallowing an ounce and a half of laudanum, and in an hour and a half as much more. Though some excitement and a slight numbness followed, he appeared so natural for seven hours that at that period his story was not credited by the medical man to whom he related it. Stupor did not set in until the *eighteenth hour;* and two hours later he was completely narcotized. After seven hours' assiduous treatment he was aroused, and eventually recovered. There seems good reason to believe that the state of alcoholic intoxication tends to postpone the time for the development of the usual symptoms. Many other cases have been reported in which this interval amounted to from five to ten hours.

Fatal period.—According to numerous observations, the ordinary duration of fatal poisoning by opium is from seven to twelve hours; although many exceptions occur in both extremes. Dr. Lyman reports a case in which an ounce of laudanum taken by a female, aged fifty-two years, produced violent symptoms in thirty-five minutes, and death in *three-quarters of an hour* (Am. Jour. Med. Sci., Oct., 1854). Dr. Coale, in the same journal, reports a case in which death took place within the same period. When a patient survives twelve hours, there is usually good hope for recovery.

On the other hand, instances are recorded in which death was delayed much beyond the usual period,—as late as twenty-four to forty-eight hours.

Fatal dose.—This cannot be stated with accuracy. An ounce of laudanum (equivalent to about forty grains of opium) has frequently proved fatal. Dr. Wormley mentions the case of a robust, healthy girl, aged seventeen years, who died in seven hours after swallowing two fluidrachms of laudanum (Micro-Chem. of Poisons, p. 460). Dr. Toogood mentions a case in which twelve drops of *Battley's Sedative*—which is believed to be two or three times as strong as common laudanum—caused death in a feeble woman, aged fifty-five years, on the day after it was taken (Prov. Med. and Surg. Jour., Nov., 1841). Sir R. Christison quotes a case where four and a half grains mixed with nine grains of camphor caused death in an adult in nine hours; and Dr. Taylor alludes to two cases, one of which proved fatal from a dose of ten grains, and the other, from eight grains.

From all that can be learned from experience, and from recorded cases, *four or five grains* may be regarded as the minimum fatal dose for an adult.

It should not be forgotten that young children are extremely susceptible to the narcotic impression of opium. Many instances might be cited where death has ensued to very young infants from a dose of one or two drops of laudanum. It should be remembered, in this connection, that old samples of laudanum, kept in bottles which are frequently opened, are considerably stronger than that which is freshly prepared, in consequence of the evaporation of the spirit. Hence it is quite possible that a single drop of laudanum taken from the dregs of a bottle might possess the strength of two or three drops of the ordinary preparation. Severe symptoms, and even death, have followed the taking of a single grain of Dover's powder by a very young infant. Trousseau states that he has seen narcotic effects in children from a dose of the wine of opium equivalent to less than the one-hundredth of a grain of this drug. It is well known that a child may be narcotized by the milk of a nurse who has taken opium. Bouchardat relates that nine new-born children were narcotized by the decoction of a single poppy-head. For further cases under this head, see Taylor "On Poisons," p. 533. This extreme susceptibility on the part of children to the action of opium should suggest caution in its administration to this class of patients.

On the other hand, recoveries are constantly taking place from very large poisonous doses of opium. Dr. Jackson reports the case of a woman who swallowed ninety grains of solid opium, and who, when seen by a physician three hours afterwards, was laboring under all the symptoms of opium-poisoning. Yet she recovered, under active treatment. (Am. Jour. Med. Sci., 1854, p. 385.) Dr. Wormley records the following remarkable case taken from the "American Medical Record," vol. xiii. p. 418. A pregnant woman, aged thirty-two years, took, suicidally, *between seven and eight ounces* of solid opium. When seen by a physician about an hour afterwards, she was able to give a connected account of the case. Prompt and repeated vomiting brought away large quantities of the solid drug. She fell into a deep sleep; but after a time reaction took place, and symptoms of phrenitis manifested themselves: finally, however, she recovered.

The following cases are taken from Wharton and Stillé's "Medical Jurisprudence," 1873, vol. ii. p. 543. A gentleman aged seventy-two years recovered from the effects of twelve drachms of laudanum. Another recovered after taking upwards of an ounce. An infant of twelve months recovered from the effects of seventy-two drops; another, six days old, after taking two grains of opium; and a child nearly six years old, from a dose of seven and a half grains.

The deleterious effects arising from the constant and ignorant use of the different nostrums everywhere sold as "soothing" potions for infants are but too well known. It is very certain that many infants annually perish from this single cause. We have witnessed an instance in which a drachm of "Winslow's Soothing Syrup" came very near proving fatal in an infant a few weeks old. Profound narcotism, with extremely contracted pupils, resulted; but the child recovered under the cautious use of the tincture of belladonna.

External application.—Opium applied to the skin, and especially to an abraded surface, or used as an injection, or introduced into the nostril or the ear, may produce dangerous, or even fatal, results. M. Tournon, of Bordeaux, relates a case in which death was attributed to four grains of opium introduced into the ear (Guy's Foren. Med., p. 505). Well-authenticated cases are reported where the application of laudanum to the sound skin has been followed by fatal coma. It is especially dangerous in the case of infants. Sir R. Christison relates two instances where the external application of laudanum proved fatal to adults. In these cases a poultice saturated with laudanum was applied over the abdomen to relieve pain: fatal narcotism followed, death taking place after some hours. In one of these cases, a strong odor of opium was exhaled from different parts of the body, on post-mortem examination, showing how completely the poison had been absorbed.

The very decided and even fatal effects of opiate enemata and suppositories are familiar to physicians. The introduction of morphia hypodermically, now so much and so advantageously employed in medicine, is sometimes attended with serious, and even fatal, results.

The influence of *idiosyncrasy* and *habit* is particularly observable in the case of opium, and is of sufficient medico-legal importance to admit of a brief notice here. Christison mentions an instance of a gentleman who was always narcotized by only seven drops of laudanum; and Taylor observed alarming symptoms from the injection of only one grain of opium. Grisolle states that he saw narcotism induced in a lady by half a grain. Every physician can recall cases where patients have been unable to take even the smallest quantity of this drug, owing to some peculiarity of constitution. Sometimes, though more rarely, there is a remarkable natural tolerance of opium, which cannot be ascribed to habit. It is more usual, however, to meet with cases like the above in the course of certain diseases. In aged persons affected with catarrh, an ordinary dose of opium may occasion alarming and even fatal consequences. Several instances of this character are given by Christison; and they should be remembered by the legal physician, since similar ones might be brought forward as instances of intentional poisoning. On the other hand, severe nervous diseases, such as tetanus and mania-a-potu, will tolerate enormous doses of opium.

Every person is aware of the effects of *habit* in modifying the tolerance of opium. Thirty, fifty, and even a hundred grains a day are taken by some opium-eaters. De Quincey, the English opium-eater, brought himself to drink *nine ounces* of laudanum, equivalent to three hundred and thirty-three grains of solid opium, a day! (See "Confessions of an Opium-Eater.")

Morbid appearances.—The only post-mortem indications of opium-poisoning are fullness of the vessels of the brain and of its sinuses. Occasionally there has been found an extravasation of serum in the ventricles and between the membranes; but very rarely of blood. Sometimes there is engorgement of the lungs, and of other vascular organs, more especially if death has been preceded by convulsions. The stomach is often perfectly natural in appearance; though at times it is somewhat reddened. But this might easily be ascribed to accidental causes. The blood is apt to be fluid. From this description it will be perceived that there is absolutely nothing in the morbid appearances that can with certainty indicate poisoning by opium. Sometimes the above signs are altogether absent; and again, when they are present, they are equally ascribable to disease. In some cases there is a strong odor of opium perceived on opening the stomach, if the poison has been swallowed in the solid or liquid state, and has not previously been evacuated by emesis. The surface of the body is usually livid; and the animal heat is said to persist for a long time, even after cadaveric rigidity has set in. All authorities unite in the opinion that it is altogether impossible to draw any positive conclusion as to opium-poisoning from the post-mortem appearances *alone.*

Treatment.—The first effort should be to remove the poison from the stomach. This can usually be accomplished by emetics, the most appropriate of which is sulphate of zinc. The dose of this should be at least double the ordinary quantity, in consequence of the torpor of the stomach. In the absence of sulphate of zinc, mustard and warm water may be advantageously employed. The emetic should be repeated, if the first dose fails to act. If emesis cannot be effected, the stomach-pump should be used, the stomach being thoroughly washed out with warm water. The next point is to overcome the constantly-increasing lethargy. For this purpose cold water should be dashed upon the face and chest; and when somewhat aroused, the patient should be kept walking about between two attendants, the tendency to relapse into stupor being counteracted by shaking him and shouting to him. When sufficiently aroused, he should be made to swallow a cup of strong coffee without sugar and cream. If these means do not succeed, electro-magnetism should be at once employed, the current being sent between the upper part of the spine and the chest. This latter means is usually very efficacious. In desperate cases, artificial respiration should be resorted to.

No *chemical* antidote can be relied upon, to the exclusion of the above-mentioned means. Tannic acid, and the iodated iodide of potassium, form insoluble precipitates with morphia, and have hence been recommended as antidotes. Belladonna, or its alkaloid atropia, is now generally regarded as a true physiological antidote to opium. Cases are every year accumulating to testify to its value and reliability; and, conversely, opium, or its alkaloid morphia, is the antidote to belladonna.

The experiments of Dr. John Harley seem to prove the reverse of the above statement, in regard to the lower animals, and that their combination rather increases the effect. Some experiments of our own on dogs, made a few years since, also go to show that there is no real antagonism between morphia and atropia in these animals. Nevertheless, judging from our own experience, as well as from the accumulated testimony of others, in reference to the human subject, we cannot withhold our conviction that they are antidotal to each other *in man.* (See papers by the author in Am. Jour. of Med. Sci., April and July, 1871.) In a case of opium-poisoning, the tincture of belladonna, or an equivalent solution of atropia, should be carefully administered in successive doses, until the pupils begin to dilate and the breathing becomes increased in frequency. Should the patient be unable to swallow, the atropia may be administered hypodermically. Out of nine cases of opium-poisoning treated by belladonna, and eighteen of belladonna-poisoning treated by opium, collected by Dr. W. F. Norris, of Philadelphia, only two of the former, and one of the latter, proved fatal. (Am. Jour. of Med. Sci., Oct., 1862, p. 395.)

MORPHIA.—Morphia, when pure, occurs in colorless, rhombic prismatic crystals; taste, very bitter. It is very slightly soluble in water; soluble in alcohol, especially when hot; slightly soluble in commercial ether; and nearly insoluble in chloroform. When ether is agitated with morphia *immediately* after the alkaloid is liberated from one of its salts by the addition of an alkali, it will dissolve a much larger proportion. *Amylic alcohol* is a rather better solvent for it than common alcohol. *Common acetic ether* is the best solvent; one part of the ether, according to Wormley, dissolving seventy-five parts of morphia. It is freely soluble in the fixed alkalies; less so in ammonia. When heated on platinum, the crystals fuse to a brownish liquid, burn like resin, evolving white fumes, and leaving a carbonaceous mass. Heated on a porcelain slab, it yields a crystalline sublimate of peculiar form. Its solution, in common with the other alkaloids, is precipitated by tannic acid. The *salts* of morphia are freely soluble in water and in diluted alcohol; but they are insoluble in ether, chloroform, amylic alcohol, and pure acetic ether.

The *symptoms* produced by morphia in the main resemble those of opium. As a rule, they manifest themselves rather earlier than in the case of the crude drug. Some have supposed that the convulsive effects occasionally witnessed after taking a poisonous dose of opium are especially due to morphia. A number of cases have been reported in which morphia, when given in poisonous doses, has occasioned decided convulsions. In the celebrated case of Dr. Castaing, a pupil of Orfila, who was tried and executed in Paris in 1823 for poisoning Auguste Ballet with morphia, the accused was proved to have recently purchased twelve grains of tartar emetic and twenty-six of acetate of morphia. The deceased had, in addition to vomiting and purging, convulsions, locked jaw, rigid spasms of the neck and abdomen, inability to swallow, and loss of sensibility in the legs. The prisoner was believed to have administered the morphia to his victim first, and afterwards the tartar emetic for the purpose of removing all traces of the former from the stomach. The suspected poison could not be detected by analysis after death.

An anomalous case of poisoning by morphia, in which the symptoms might have raised the suspicion of strychnia, although there were obvious points of difference, is reported by Dr. Ferris in the "British Medical Journal," November 11, 1871. A woman aged sixty years took twenty-five drops of a solution of morphia for a bad cough and diarrhœa. The strength of the solution is not given; though it is stated that it was poured from the shop-bottle of a druggist, labeled "ten to sixty drops for a dose." Two hours after, she was found bathed in perspiration, face swollen and eyeballs protruding. Both sides of her mouth were twitching; the arms were bent and moved backwards and forwards convulsively. In a moment the convulsions ceased, and she complained of great pain in the chest and was unable to take a long breath. After a time the convulsions returned, and there was great pain in the back, and partial opisthotonos. When seen by the physician, four hours after taking the morphia, she complained of great pain in the chest and bowels; the breathing was short and quick; there was twitching in the legs and down the spine, together with pain in this region. The pupils were extremely contracted. There never was any disposition to sleep. There was an entire absence of trismus. The convulsive movements recurred several times after a complete cessation. The patient gradually recovered; her last symptoms being slight sickness, pain in the head, and contracted pupils.

The salts of morphia most used are the *sulphate* in this country, and the *hydrochlorate* and *acetate* in Great Britain. These are usually estimated to have about six times the strength of opium.

Fatal dose.—Dr. Taylor (Prin. and Prac. of Med. Jurisp., 1873, vol. ii. p. 358) mentions four recorded cases in which *one grain* of hydrochlorate of morphia proved fatal to adults: in one, in solution; in the second, in pill; in the third, in powder; in the fourth, by hypodermic injection. In the first of these cases, the morphia was taken in divided doses in six hours: the patient died in about seven hours. The second case died in thirteen hours, the symptoms coming on in three hours: no morphia was discovered in the stomach after death. In the third case, death ensued in ten hours, the symptoms appearing in about three hours. In the fourth instance, one grain of morphia was administered hypodermically in three divided doses, all within ten hours. The man slept quietly for two hours; he then took dinner, and engaged in conversation; but in another hour he suddenly became insensible, and died two hours after, in profound narcotism. We have known the case of a gentleman in whom about *three-quarters of a grain* taken hypodermically proved fatal within twenty-four hours.

On the other hand, enormous doses of this alkaloid have been swallowed without fatal consequences, and quite independently of the effect of habit. One of the most remarkable cases yet reported is related by Dr. W. F. Norris (Amer. Jour. Med. Sci., October, 1862, p. 395). A druggist aged nineteen years, for the purpose of self-destruction, swallowed *seventy-five* grains of sulphate of morphia. No marked symptoms appeared for an hour and a half afterwards, when he began to feel sleepy, and had a staggering gait. Soon after this, emetics were given, causing free emesis. He then became unconscious; the pupils contracted to a point; the pulse was soft and frequent; respiration slow and labored; but under the active use of remedies, including extract of belladonna, galvanism, and the cold douche, he was quite well on the second day after the occurrence.

The external application of morphia to abraded surfaces, as also in the form of enema, has sometimes been attended with fatal effect. One grain sprinkled over a blistered surface on the back of an aged lady produced, in about two hours, the most alarming symptoms, from which she barely recovered. Much smaller doses than this have occasioned very serious symptoms. Dr. Anstie met with a case in which *three grains* given by enema caused death in sixteen hours.

There are no characteristic *post-mortem appearances* to indicate death from morphia. There may be, as in the case of opium, fullness of the cerebral vessels, with serous effusion, and bloody points on section of the brain-substance. It produces no local irritant action on the stomach and bowels.

Chemical analysis of opium.—As opium is a very complex substance, there are no chemical tests for it *as such:* it can be identified by its physical properties of odor and taste, and by its action on living animals. As the peculiar odor depends on a volatile principle, it may soon disappear after exposure of the material to the air, or on heating it. Again, it may be concealed by other odors, or it may be destroyed in consequence of some organic change undergone by the material in the stomach before death. The only means of identifying the opium in a case of suspected poisoning, is to detect its contained *meconate of morphia*, and especially its *meconic acid.* The identification of this latter ingredient is absolute proof of the presence of opium or of some of its preparations, since it is found exclusively in opium. In poisoning by morphia or its ordinary salts (except the meconate), the meconic acid is, of course, always absent.

Detection of morphia.—The chemical tests are best applied to the morphia in the *solid* state. (1) Concentrated nitric acid dropped upon a fragment produces a rich orange-red color, and dissolves it with effervescence, and the production of ruddy fumes of nitrous acid. The orange-red solution slowly fades to a yellow. If the morphia be in solution, the acid should be in large excess: in this case the color is lighter than when the morphia is in the solid state. Nitric acid also strikes a red color with *brucia*, which, on the addition of protochloride of tin, changes to a bright purple; whereas no change is produced in the case of morphia. (2) Strong sulphuric acid slowly dissolves it without change of color: if now a crystal of bichromate of potassa be stirred in the solution, it slowly acquires a green color, from the production of the oxide of chrome. If heat be applied to the original sulphuric acid solution, it assumes a brown color. Prof. Otto says that a very minute quantity may be detected by first dissolving it in a drop or two of concentrated sulphuric acid by the aid of heat; after cooling, dilute with a little water, and add a crystal of chromate of potassa; the liquid acquires an intensely mahogany-brown color. (3) A drop of a solution of a salt of morphia (as the acetate), exposed for a few moments to the vapors of ammonia, will deposit the alkaloid in the form of prismatic or hexagonal crystals, easily distinguished under the microscope. (4) Neutral sesquichloride or persulphate of iron dropped on a crystal of morphia renders it deep blue,—turning to green, if added in excess. For the success of this experiment it is indispensable that no free acid be present. Tardieu (Sur l'Empoisonnement, p. 879) gives the following formula for properly preparing the solution of persulphate of iron. Introduce into a small receiver a mixture of one part of pure sulphuric acid and one and a half parts of distilled water, saturated by an excess of sesquioxide of iron, at the temperature of a water-bath. When the liquid has become saturated with the oxide, it is filtered, and the clear filtrate is properly preserved. An excess of acid, a degree of heat above 122° F., and the presence of organic matter will prevent the production of the blue color. (5) *Iodic acid.*—Dissolve a small quantity of this acid in a drop of cold, freshly-made starch, place it on a white slab, and add the fragment of morphia. Iodine is liberated, and immediately imparts the characteristic blue color to the starch. M. Dupré advises that the morphia or its solution be first treated with a drop of the starch-solution; the mixture is then carefully evaporated to dryness, and the residue, after cooling, moistened with a solution of iodic acid. In this manner a residue containing only one ten-thousandth of a grain of the alkaloid will yield a distinct blue color (Wormley). (6) Put a small fragment on a porcelain slab, with a glass disk properly supported over it, and apply a heat of about 330° F.: a crystalline, feathery sublimate is formed, of peculiar appearance, and varying according to the degree of heat. The one-hundredth of a grain will thus yield highly satisfactory results, easily visible by the microscope. (Guy's Forens. Med., p. 499.)

(7) *Sulpho-molybdic acid.*—This is made by dissolving with a gentle heat five or six grains of powdered molybdate of ammonia in two drachms of strong sulphuric acid. The liquid should be freshly prepared, and kept from contact with air and organic matter. When one or two drops are rubbed with dry morphia, or any of its salts, an intense purplish or crimson color is produced; this changes to a dingy green, and ultimately to a splendid sapphire-blue. A very minute trace of morphia is thus revealed. This test produces no change on strychnia, the mixture slowly acquiring a pale-blue tint. On brucia and veratria it ultimately produces a dark-blue color. The action on salicine closely resembles that produced on morphia. (Taylor's Med. Jurisp., Am. ed., 1873, p. 210.)

(8) *Iodic acid with sulphide of carbon* (Taylor).—A solution of iodic acid should be mixed with its volume of sulphide of carbon: there ought to be no change of color. On adding a small quantity of the mixture to morphia or its salts, either solid or in solution, the iodine is liberated and is dissolved by the sulphide, which sinks to the bottom, acquiring a pink or red color, varying in intensity according to the quantity of morphia present. The presence of morphia may thus (according to Dr. Taylor) be distinguished in one drop of laudanum, in chlorodyne, and other liquids, in spite of the presence of organic matter.

Several other tests are mentioned in the books, but they are not so characteristic as those above described. These are *iodide of potassium and iodine*, *terchloride of gold*, *bromine in hydrobromic acid*, etc.

Meconic Acid.—This acid is pecular to opium, in which it occurs in combination with morphia. Its detection, therefore, in a suspected material may be regarded as positive proof of the presence of opium. In its pure state, it is in the form of colorless crystalline plates, tolerably soluble in water, especially if hot; more so in alcohol. It is believed to be inert in the human system, judging from experiments made upon animals.

Tests.—(1) By far the most delicate chemical test is *sesquichloride* or *persulphate of iron*, each of which strikes with solutions of meconic acid, and also with the acid in its solid form, a blood-red color, which is not removed by dilute acids, by corrosive sublimate, or by chloride of gold, but is discharged by protochloride of tin and sulphurous acid. This test succeeds even in very dilute solutions of meconic acid; and it is owing to the presence of this acid that a minute quantity of laudanum, or of other liquid preparations of opium, diffused in a large quantity of water, will yield a red color on the addition of a persalt of iron. The only fallacy that would be likely to occur in a medico-legal case is from the presence of some *sulphocyanide* in the material examined, as the sulphocyanide of potassium of the saliva, which yields a similar red color with a persalt of iron. We have frequently verified the experiment with human saliva. Of course, if the contents of the human stomach (which must necessarily contain more or less saliva that has been swallowed) be subjected to the above test, in a suspected case of opium-poisoning, an erroneous inference might be drawn if this single test were relied on: it should always be supplemented by the addition of a solution of corrosive sublimate, which instantly dissolves the red sulphocyanide of iron, but has no effect on the meconate.

Another fallacy, although less likely to occur than the foregoing, is that occasioned by strong *acetic acid* or its salts, both of which give a red color with persalt of iron; and this color, moreover, is not removed by corrosive sublimate or chloride of gold. But if previously boiled with dilute sulphuric acid, the acetate gives no color with the iron salt, and is thus distinguished from a meconate. Again, an acetate solution differs from a meconate in not yielding any precipitate with acetate of lead. A concentrated solution of *white mustard* also imparts a red color to a persalt of iron, which, however, is immediately removed by corrosive sublimate, but not by chloride of gold.

(2) *Acetate of lead* yields a yellowish-white precipitate—meconate of lead, insoluble in an excess of acetic acid. This reagent is thus employed to separate the meconic acid in the analysis of a case of opium-poisoning. (3) *Chloride of barium* yields a white, crystalline deposit of a peculiar form. (4) *Nitrate of silver* produces an amorphous precipitate of a yellowish or white color, which becomes red on the addition of a persalt of iron. Other tests are *ferrocyanide of potassium*, *chloride of calcium*, and *hydrochloric acid.*

Although the other alkaloidal constituents of opium are not usually sought for in cases of poisoning by that drug, it may be proper here to allude to these very briefly, mentioning the characteristic tests for each.

NARCOTINA.—This alkaloid, when pure, occurs in colorless, transparent crystals, or as a granular powder. It has a bitter taste; is insoluble in water, but readily soluble in alcohol and ether, and still more so in chloroform. Narcotina is usually considered inert. Orfila found that thirty grains were required to kill a dog; and, according to Gmelin (Handbook of Chemistry, xvi. p. 137), doses of one hundred and twenty grains are said to have been given to man without effect. Dr. S. Weir Mitchell, of Philadelphia, took thirty grains, with no appreciable result (Effect of Opium and its Derivative Alkaloids: Am. Jour. of Med. Sci., Jan., 1870, p. 23); but he found, by an unexplained anomaly, that this substance acts, in doses of two to seven grains, as a powerful convulsant on pigeons, terminating in speedy death. This effect contrasts remarkably with that of morphia, of which enormous doses are required by birds in order to produce effect. Both alkaloids excite in them spasms, which are frequently tetanic in character. Neither of them produces stupor.

Unlike morphia, narcotina produces no color with a persalt of iron or a mixture of iodic acid and starch. Its characteristic *test* is the action of sulphuric acid and nitrate of potassa.

If a minute fragment of narcotina, or the deposit resulting from evaporating a small portion of the solution, be dissolved in a few drops of strong sulphuric acid, and a small crystal of nitre be stirred in the solution, it will quickly acquire a deep blood-red color. This color disappears on adding an excess of nitric acid.

Codeia.—This alkaloid has much stronger basic properties than narcotina. It occurs in white crystals; it is quite soluble in water, alcohol, ether, and chloroform. Its taste is bitter. Codeia is regarded by Magendie as a hypnotic and stupefier. Drs. Harley and S. W. Mitchell deem it to possess but feeble hypnotic powers. In man, it first causes excitement, with contracted pupil, slight giddiness, and some gastro-intestinal derangement. On birds, however, Dr. Mitchell found that it acted as a more powerful convulsant than narcotina; death occurring from smaller doses and in a shorter time. It differs from morphia in not decomposing iodic acid, and in not being reddened by nitric acid. From narcotina it is distinguished by not being turned red by sulphuric acid and nitrate of potassa. *Nitric acid and potassa* form one of its best tests. When strong nitric acid is added to a small quantity of codeia, it dissolves it, with the evolution of nitrous fumes, and on evaporation leaves a yellowish residue. On touching this with a drop of caustic potassa, it assumes a beautiful orange tint.

Narceine.—This is usually considered to be a neutral principle. It exists in opium in about the same proportion as morphia. Narceine is regarded by M. Bernard as a hypnotic of even greater power than morphia; but this idea does not seem to be supported by the experiments of Harley and Mitchell. The latter was not sensible of any soporific effect after taking a dose of five grains. The same is also true of birds (*loc. cit.*, p. 26). It is not acted upon by a persalt of iron. It crystallizes in delicate, needle-shaped tufts. It is very insoluble in water; strong sulphuric acid gives it a reddish-brown color, which, on the application of heat, changes to a deep red. When dropped into concentrated hydrochloric acid, it becomes blue, and dissolves into a colorless solution. All samples will not yield this blue color when treated as above.

THEBAIA, or PARAMORPHIA, is another of the alkaloids found in opium. Its tetanizing power has long been known,—a dog, according to Dr. Harley, being killed by two grains given hypodermically. In Dr. S. W. Mitchell's experiments it proved speedily fatal to pigeons, exciting in them the most violent tetanic convulsions, closely resembling those of strychnia, in the course of a minute or two, and with only one-third of a grain given hypodermically. In fact, of all the principles of opium, this substance was found to be the most powerful toxic to birds.

MECONIN, or OPIANYL, is a neutral crystalline substance occurring in delicate needles or in long prisms; taste, somewhat bitter. Strong sulphuric acid dissolves it to a colorless solution, which, on being heated, becomes of a beautiful blue or purple color. A very minute portion will yield satisfactory results with this reagent.

Detection of opium in organic mixtures, or in the contents of the stomach.—It occasionally happens that the strong odor of opium can be recognized in organic mixtures, as *e.g.* in the matters vomited, or in the contents of the stomach after death: this, of course, will assist very materially in the diagnosis of the case. As a rule, however, this aid will not be furnished, and when we come to apply the characteristic chemical test to the stomach after death we shall fail to recognize the poison sought, in consequence of its removal by vomiting, or by digestion, decomposition, or absorption. Especially is this true in the case of infants who have perished after a very small dose,—*e.g.* from a few drops of laudanum. The highest authorities unite in the opinion that in cases of poisoning by crude opium or its liquid preparations it is the exception, and not the rule, for the analyst to be able to recognize it in the stomach after death. (See Christison and Taylor on Poisons, art. *Opium.*) It is much more likely to be found in the matters vomited. In any case, the proper mode of proceeding is to cut up the solid matters, if any, into very small pieces, adding distilled water, if necessary, with a little alcohol, acidulating with pure acetic acid; the materials should be well mixed together, and subjected to a gentle heat. A trial test should now be made of a portion of the clear liquid with nitric acid and a persalt of iron for morphia and meconic acid respectively. If the liquid be highly colored, it will be advisable to dilute it with water before applying these tests. It will be remembered that the iron test will detect a very minute quantity of meconic acid.

After heating the mixture, as above described, for about half an hour, it should, after cooling, be strained through muslin, the solid residue well washed with strong alcohol, and pressed, the washings being added to the first liquid. The liquid is next to be evaporated over a water-bath to a small volume; and, when cooled, it is to be filtered through filtering-paper previously wetted. The filter is to be washed with dilute alcohol, and the washings added to the filtrate. To the clear filtrate acetate of lead is added in slight excess: this precipitates the meconic acid as *meconate of lead*, and leaves the morphia in solution as an *acetate*. Any sulphocyanide present in the stomach would also remain dissolved. When the precipitate has completely subsided, the whole is transferred to a moistened filter, and the solid residue is completely washed with distilled water.

(*a*) The impure meconate of lead on the filter is to be washed into a deep glass, by piercing the filter and using a wash-bottle. A stream of sulphuretted hydrogen is now passed through the water containing the diffused meconate, as long as any sulphide of lead is thrown down. The liberated meconic acid will remain in solution. The liquid is now filtered, and the clear filtrate is concentrated by a gentle heat, which also serves to expel the excess of sulphuretted hydrogen. A few drops of the solution may now be examined by a persalt of iron, and if the presence of meconic acid is decidedly indicated, the remainder of the liquid may be tested with the other proper reagents. On proper concentration, the liquid will yield crystals of meconic acid, provided this be present in sufficient quantity. If, however, little or no indication of meconic acid is given, the liquid should be carefully concentrated to a small volume, and a drop or two should be tested with the iron and other tests, before deciding on the absence of the poison.

As the presence of foreign matters interferes with the delicacy of these operations, it may be advisable either to evaporate the liquid to complete dryness, and dissolve the residue in warm water and filter, or to reprecipitate with acetate of lead, and decompose, as before, with sulphuretted hydrogen.

Another method for decomposing the meconate of lead is to digest it at a moderate heat with dilute sulphuric acid, which throws down the lead as an insoluble *sulphate*, and leaves the meconic acid in solution. The former process is, however, on the whole, to be preferred.

(*b*) The original filtrate, it will be remembered, contains all the morphia in the form of an acetate, together with an excess of acetate of lead. It should be treated with sulphuretted hydrogen until all the lead is precipitated, then filtered, and the filtrate concentrated by gentle heat to dryness. The residue should be dissolved in a little warm, distilled water, and a drop or two examined for morphia with nitric acid and perchloride of iron, as trial tests. Whether or not these tests indicate the presence of morphia, the remaining liquid, diluted with distilled water, if necessary, is made distinctly alkaline by carbonate of potassa, and allowed to stand for some time; then it is to be agitated with several volumes of *absolute ether*. Under these circumstances none of the morphia is taken up by the ether, which only separates certain foreign matters. The ethereal solution is to be carefully decanted, and set aside for future examination. The remaining alkaline liquid is now to be shaken violently with four or five times its volume of a mixture consisting of two parts of absolute ether and one of pure alcohol. This operation is best performed in a long glass tube. By this process the morphia is separated, and will be contained in the supernatant ethereal mixture. On carefully decanting this, and allowing it to evaporate spontaneously in a watch-glass, the morphia will often be left in a crystalline form; but if it exists in very minute quantity, and is mixed with much foreign matter, the deposit will be amorphous. In the latter case, Prof. Wormley advises to wash the dry residue by gently rotating a few drops of pure water over it in the glass, and then decanting the liquid. Small portions of the solid residue are now to be tested by nitric acid and perchloride of iron, and the remaining portion of the residue is dissolved in a few drops of water, with the aid of sufficient acetic acid. This solution may then be subjected to the various liquid tests (*ante*, p. 375). A few drops of this solution, if exposed to the vapors of ammonia, will become alkaline, and, after some hours, will deposit the alkaloid in its characteristic crystalline form, as shown by the microscope.

The process of Uslar and Erdmann is recommended by good authorities as of equal, if not superior, value to the one above described. By this method, *amylic alcohol* is employed to dissolve out the morphia from the alkaline solution, instead of ether and alcohol. Two or three volumes of hot amylic alcohol are shaken up with the liquid, and, after being allowed to rest for some time, the upper or alcoholic stratum is to be removed by means of a caoutchouc pipette, and allowed to evaporate to dryness in a watch-glass by the aid of a gentle heat: sometimes the morphia may be recovered by this means in the crystalline form, though it is more likely to be left in an amorphous state than when deposited from an ethereal solution. Should the residue be amorphous, it may be redissolved in a small quantity of ordinary dilute alcohol, and allowed to evaporate spontaneously.

Examination for morphia alone, or its salts.—When there is reason to believe that the poisoning has been occasioned by one of the salts of morphia, the same method of analysis of the suspected material is to be followed, with the omission of the acetate of lead and sulphuretted hydrogen,—inasmuch as no meconic acid is present. The ultimate extraction of the alkaloid may be effected by the alcohol-ether, or by the amylic alcohol.

Detection in the tissues and blood.—Thus far there has been an almost entire failure to detect the poison in any of the organs of the body. Prof. Wormley succeeded in showing the presence of both morphia and meconic acid in the *blood* of several dogs and cats, by the nitric acid, ammonia, and iron tests. There is considerable doubt about the detection of these principles in the *urine*, inasmuch as the results alleged to have been produced by certain reagents, and supposed to indicate the presence of morphia or meconic acid, have since been proved to be due to substances existing normally in the urine.

As there is no medicine more freely prescribed than opium and its preparations, the discovery of mere traces in the stomach after death should not be considered as *of itself* indicating death from poison.

Before taking leave of this subject, it is proper to guard the analyst against a too hasty conclusion as to the presence of opium or its alkaloid, in a medico-legal case, *derived from the color alone.* In a preceding chapter (p. 77) we have taken occasion to point out the dangerous fallacy of relying merely on the *color* of different tests in a medico-legal examination. Orfila tells us that MM. Ruspini and Cogrossi found that a decoction of a calf's intestines, although no morphia was present, acted on iodic acid like that alkaloid (Toxicol., ii. p. 232). In another case morphia was pronounced to be present in urine by reason of the action of the extract of this liquid on iodic acid. The effect was found to be due to uric acid and urate of ammonia. Dr. Taylor cites a very instructive case in which sudden death from apoplexy was imputed to opium, and where a decoction of the contents of the stomach of the deceased, treated with nitric acid, yielded a red color, similar to that produced by morphia; but no morphia was separated, and no meconic acid found, or even sought for. Upon this very questionable result, a distinguished chemist (?) who made the analysis deposed at the inquest that he had found in the stomach "distinct traces of morphia," and that the quantity discovered was about the tenth of a grain; and the smallness of the quantity was ascribed to absorption. The facts which subsequently transpired showed that there could have been no morphia or opium present in the stomach; but that the person had died from purely natural causes. The author quoted further justly remarks, that "unless morphia or meconic acid, or both, have been separated and obtained from the suspected liquid, no man is justified in swearing that opium is present, and still less that it has been the cause of death. The correspondence on this subject conveys an important lesson to medical witnesses, and especially to the 'experts' in the chemical branch of medical jurisprudence." In the above case it was fortunate that some of the medicine last taken by the deceased remained, so that the absence of morphia was proved by analysis; otherwise the husband who administered it, or the druggist who compounded it, might have been charged with manslaughter, especially as the symptoms (apoplexy) resembled those of opium-poisoning, and this was further supported *by the oath of the* " *expert.*"

SECTION II.

POISONING BY ALCOHOL.

The poisonous effects of Alcohol are of a twofold character: 1, those with which we are most familiar as the result of habitual intoxication, as witnessed in common drunkards; and 2, those which follow the ingestion of a single very large dose of alcoholic spirits. The latter only will be considered under the present head. Instances of this kind of poisoning are most commonly witnessed in those silly and criminal cases where for a wager, or as a mere bravado, a large quantity of spirits is drunk off at one time, and also in cases of young children who have accidentally swallowed, or who have been made to take, a large draught of some strong alcoholic beverage. In both cases, it is to be observed, the poisonous and fatal effects are produced not so much upon persons who are accustomed to the use of alcohol, as upon those who are not thus habituated.

The symptoms of this *acute* alcoholic poisoning are well marked. The individual, after swallowing the large potation, almost immediately falls into a profound coma. There is little or none of the previous excitement which is usually so characteristic of the early stages of common alcoholic intoxication. There is a vacant, ghastly expression of the features, which are sometimes suffused or bloated; the lips are livid; the pupils dilated and fixed; the conjunctivæ are suffused; an alcoholic exhalation from the breath is recognized; there are apt to be convulsive movements of the limbs; respiration, at first stertorous, becomes more and more difficult; a bloody froth appears on the lips; involuntary evacuations occur; and death ensues sometimes in half an hour, or even earlier, after the fatal drink (Tardieu). In other instances, the individual may apparently recover from the first effects, and then suddenly become insensible, and die in convulsions.

If the dose has not been so overpowering, other symptoms may precede the profound coma, such as headache, giddiness, confusion of mind, loss of muscular power, staggering gait, stammering speech, and slight convulsive movements. Recovery may take place after a prolonged sleep, or sooner, if vomiting occurs. The very large quantities seem to destroy life by shock.

Post-mortem appearances.—A remarkable absence of putrefaction of the body is generally observed. There is usually intense congestion of the stomach, as indicated by a deep-red color, either diffused or in patches. More or less congestion of the brain and lungs may always be expected, together with serous effusion. Tardieu mentions a case where clotted and thickened blood was found in the cavity of the arachnoid, and also in the lungs (*op. cit.*, p. 847). Usually a strong odor of alcohol can be perceived in the different tissues of the body; but the organs for which this poisonous fluid displays the greatest affinity are the brain and liver, and, accordingly, it is from these organs, especially, that we may expect to recover the alcohol, after death, by distillation. If life has been protracted for several hours, all traces of the odor may have disappeared both from the stomach and from the other organs; though it is more permanent in the latter.

The *diagnosis* of a case of acute alcoholism is generally sufficiently easy. The odor, when it exists, will serve to distinguish it from opium-poisoning. In the latter the pupils are nearly always contracted; while in the former they are dilated. Cases may occur where, in the absence of the characteristic odor, and from an ignorance of the attending circumstances, it might be difficult to distinguish between the symptoms and those of concussion of the brain. In such cases, the autopsy may throw some light upon the matter.

Chemical analysis.—Alcohol may usually be recovered after death, by distilling the stomach and its contents, provided the case has not been protracted so long as to have permitted the poison to escape by absorption. In the latter case, the attempt should be made to separate it from the brain and liver, by the same process. The whole of the suspected material should be put into a capacious retort, and distilled on a water-bath, with a proper condensing apparatus. If it has an acid reaction, it should first be neutralized by the addition of carbonate of potassa or soda. The distillate should be mixed with chloride of calcium or quicklime, and distilled a second time. The second distillate is to be shaken with an excess of carbonate of potassa, and allowed to stand. The stratum of alcohol which will after a time rise to the surface may be separated by a pipette, and submitted to the following tests: (1) Its *taste* is hot and pungent; its *odor* characteristic. (2) It burns with a pale-blue flame, leaving no carbonaceous residue; the products of its combustion are carbonic acid and water: the former will cause a milky appearance in lime-water. (3) It dissolves camphor. (4) Add to it some solution of bichromate of potassa and sulphuric acid: the green oxide of chromium is developed, along with the peculiar odor of *aldehyde.* This latter test is very delicate, and will serve to detect an amount of alcohol too minute for the other processes. In this case, the experiment should be performed by moistening a few fibres of asbestos with a mixture of a strong solution of bichromate of potassa and sulphuric acid; they are then to be placed in the tube connected with the retort in which the distillation is going on: the smallest portion of alcoholic vapor coming in contact with the moistened asbestos will be indicated by the production of the green coloration due to the oxide of chromium.

Both ether and pyroxylic spirit will produce this last effect, and likewise give most of the results of alcohol. Ether may be distinguished by its *odor*, and by the yellow color of its flame, as also by its smoky deposit on porcelain. Pyroxylic spirit may be recognized by its peculiar odor, and by its smoky flame on burning.

In the tissues.—The proof of the absorption of alcohol is afforded in its detection in the blood and urine, and in the different tissues of the body. It has been frequently separated from the brain and liver by distillation. Dr. Percy detected it in the liver, blood, urine, and bile. An exceedingly delicate process has been devised by Buchheim for detecting it in small quantity in the blood or tissues, based on the conversion of the vapor of alcohol into aldehyde and acetic acid, when it is passed over platinum-black, contained in a platinum tray. As much as possible of the material, neutralized first by carbonate of potassa, should be introduced into a capacious retort, along with a little water, and distilled on a water-bath. The neck of the retort should be slightly inclined, and wide enough to hold a platinum tray about two inches long and half an inch wide, containing the platinum-black. Hanging over each end of the tray is placed a slip of moistened litmus-paper, and touching the platinum-black. The tray is now pushed forward towards the body of the retort. As soon as the vaporization of any alcohol that may be present occurs, it will be manifested by the reddening of the litmus-paper at the farther end of the tray, in consequence of the production of acetic acid, while the paper nearer to the body of the retort will remain blue. If no reddening of the paper occurs, no alcohol can be present; if the reddening rapidly occurs, the tray should be removed, and the vapor should be condensed in the usual way.

As both ether and wood spirit produce a similar effect with platinum-black, this process offers no advantage over the chromic process above described, except when putrefaction has taken place, and sulphuretted hydrogen is evolved: this would reduce chromic acid, but it does not affect the platinum-black. (Taylor on Poisons, p. 643.)

A new test for alcohol is given by Lieben (Phar. Jour., 1869). A few grains of *iodine* and a few drops of a solution of *caustic soda* are introduced into a test-tube along with the suspected fluid: it is then heated without boiling, when *iodoform* is precipitated. It is stated that one part of alcohol in two thousand of mixture can thus be detected; also, that it may thus be discovered in the *urine* after drinking, by first distilling it.

CHAPTER XXIII.

ANÆSTHETICS.

THIS subdivision of the *Cerebral Neurotics* comprises those substances which display their power chiefly by producing insensibility to pain. The only anæsthetics noticed here are Ether and Chloroform. Under this head it will also be convenient to speak of Hydrate of Chloral, although its action on the system differs somewhat from that of the others.

SECTION I.

POISONING BY ETHER.

The term *ether* is used here to designate the substance procured by the distillation of sulphuric acid and alcohol, and known generally as *sulphuric ether*. It is a limpid, colorless liquid, of a peculiar odor, and hot, pungent taste; highly volatile and inflammable; sp. gr., 0.735; boils at 95° F.; it burns with a bright-yellow flame, and deposits carbon on a cold porcelain surface. It is sparingly soluble in water, and very soluble in alcohol.

Symptoms. — Swallowed in large doses, it produces very nearly the same effects as a large amount of alcohol. There is usually a short period of delirious excitement, which is followed by coma and other symptoms of narcotism, similar to those caused by alcohol.

Morbid lesions.—From its less solubility in water, ether is a more powerful local irritant than alcohol. In the body of a dog poisoned by ether, the stomach was found to be violently inflamed, as also the mucous lining of the duodenum, though in a less degree. The heart contained black blood, partly coagulated; the lungs were gorged with fluid blood. (Orfila, Toxicologie, ii. p. 531.)

The inhalation of the vapor of ether is extensively employed, as is well known, as an *anæsthetic* agent. When thus breathed into the lungs, it is so rapidly carried into the circulation that its effects upon the brain are much more prompt and decided than when it is swallowed. Although it has produced fatal effects in a number of instances when thus employed, either through want of proper caution, or owing to some constitutional peculiarity of the patient, this result has been far less frequent than in the case of chloroform. The immediate operation of ether, when inhaled, is that of a stimulant, as denoted by a quickened pulse, flushed face, suffused eye, and mental excitement. This is soon followed, if the dose be sufficient, by a state of stupor and complete insensibility to pain. This last condition may be prolonged for a considerable time by continuing the inhalation. There is occasionally a departure from the above effects: thus, there may be violent excitement at one time; at other times, a state of incoherence; and again, nausea and vomiting.

Chemical analysis.—Ether is recognized by its taste and smell; by its combustion; by its volatility; and by its action on a mixture of sulphuric acid and bichromate of potassa—the same as in the case of alcohol.

From *organic mixtures* it is recovered by the same process of distillation as for alcohol.

SECTION II.

POISONING BY CHLOROFORM.—HYDRATE OF CHLORAL.

Chloroform is a colorless, limpid liquid, very volatile, giving off a dense vapor; sp. gr., 1.497; boiling at 142° F. It has a characteristic, agreeable odor, and a sweet, pungent taste. It is very soluble in ether and alcohol; nearly insoluble in water, in which it sinks in globules. It is not inflammable, like ether and alcohol. It is a powerful solvent of many organic substances, among which the *alkaloids* deserve special notice.

At a red heat, its vapor is decomposed into chlorine and hydrochloric acid.

Effects and Symptoms.—Chloroform, like ether, has obtained much notoriety as a powerful anæsthetic agent. This impression is more rapidly produced by it than by ether. There is, moreover, an absence of the preceding excitement that is witnessed in ether, and the patient almost immediately loses his sensibility, while the muscular system becomes completely relaxed. It appears to act as a depressant to the system from the first; and if administered in the concentrated state, and not properly diluted with atmospheric air, it may produce very rapidly fatal effects. In some instances death has occurred within two minutes from the commencement of inhalation. In one case the fatal result took place in *one minute* after breathing only thirty drops in the state of vapor; and in another case only fifteen drops proved fatal in a very short time. Occasionally, death has occurred even after the withdrawal of the vapor, apparently by its cumulative effects on the system. It is undoubtedly a far more dangerous anæsthetic agent than ether; and instances of its fatal effects are constantly being reported in the medical journals. Certain constitutional disorders render a patient particularly liable to danger from the inhalation of chloroform: among these may be especially mentioned fatty degeneration of the heart. The immediate cause of death from chloroform vapor appears to be, in the majority of cases, syncope, or a cessation of the heart's action; and in others, asphyxia.

The effects of chloroform, when taken *internally*, particularly claim the attention of the toxicologist. These effects are those of a local irritant to the stomach, together with those of a powerful narcotic, causing speedy insensibility, stupor, convulsions, dilated pupils, flushed face, a full and oppressed pulse, and foaming at the mouth. Other cases have been reported in which the pupil was contracted.

When swallowed, chloroform does not act with nearly the activity and speed that it exhibits when breathed into the lungs. It has frequently been given in medical practice in doses of one or two drachms with impunity; but many cases are reported where it produced alarming and even fatal symptoms in doses of half an ounce and upwards. The smallest fatal dose yet recorded is mentioned by Dr. Taylor (On Poisons, p. 652), where a boy, aged four years, died in about three hours after swallowing *one drachm* of chloroform. He soon fell into a state of complete insensibility, with coldness of skin and failure of pulse, at first, but subsequent reaction and stertorous breathing followed, without restoration to consciousness.

Post-mortem appearances.—In death from *liquid* chloroform, the characteristic odor will usually be apparent, together with a slow putrefaction of the body, and persistent rigor mortis. In several fatal cases reported, there was evidence of great irritation of the lining membrane of the stomach, proceeding to actual softening, and in one case also to ulceration.

In cases of death resulting from chloroform-*vapor*, the most common lesions are great congestion of the lungs and bronchial tubes, with a dark and fluid condition of the blood. Sometimes there is congestion of the vessels of the brain; but very often nothing whatever is discovered after death to indicate the manner in which this was produced.

Treatment.—In poisoning by liquid chloroform, the stomach-pump should be immediately employed. When the *vapor* has caused the danger, the chloroform should be immediately withdrawn from the nose, and the patient freely exposed to a current of air; cold affusion should be practiced; the tongue should be drawn out of the mouth to facilitate respiration; artificial respiration, and the direct electrical current, may also be employed.

Chemical analysis.—Organic mixtures, as the contents of the stomach (if they have not been too long exposed to the air), will readily yield any contained chloroform by distillation over a water-bath. The first distillate may be distilled a second time along with chloride of calcium; and the product subjected to the proper tests for chloroform,—*taste*, *smell*, *solubility*, *volatility*, etc. To recover it from the blood and tissues, in cases of inhalation, the process devised by Duroy is recommended. The blood, liver, brain, etc., finely divided, with the addition of distilled water, if necessary, are put into a large glass flask, the neck of which is fitted with a cork perforated to contain a hard glass tube bent at right angles, and from twelve to fifteen inches long. The flask is gradually heated in water to 140° F., and at the same time the middle of the tube is heated red-hot by a gas-jet. At a red heat, chloroform-vapor is decomposed into chlorine and hydrochloric acid. A slip of moistened litmus-paper placed at the mouth of the tube will be first reddened, and then bleached. Starch-paper wetted with iodide of potassium is rendered blue; and nitrate of silver solution is whitened by the production of chloride of silver, which may easily be identified. In cases of great delicacy, it will be advisable to use a porcelain retort, with a tube inserted through the tubulure, which communicates with a bellows: by this means a regulated current of air can be kept up so as to force the chloroform-vapor through the neck of the retort, which is kept at a full red heat by means of a small furnace. To the extremity of this neck are attached the Liebig's bulbs, containing a solution of nitrate of silver. Any chloroform-vapor arising from the contents of the retort is forced over the heated neck, where it suffers decomposition, and the chlorine and hydrochloric acid resulting are indicated by the white precipitate in the bulbs. The absence of any *free* hydrochloric acid in the original material should always first be insured by the addition of carbonate of soda or potash.

It is important to remember that if hydrate of chloral had been taken by the patient just previous to death, and the alkali be added to the tissues, the chloral would be decomposed into chloroform, and produce all the above reactions.

Certain important medico-legal points connected with chloroform in its anæsthetic relations, and especially the question "whether chloroform can be used to facilitate robbery," will be found ably discussed by Dr. Stephen Rogers, of New York, in the "Journal of Physiological Medicine," October, 1871. Dr. H. C. Wood states (Therapeutics, p. 253) that experiments made at the Philadelphia Hospital have proven that persons in a *sound* sleep may be chloroformed without their being awakened; but this cannot be produced on a person partially awake, or even sleeping lightly, without his knowledge.

HYDRATE OF CHLORAL.—This is a solid, crystalline substance, procured by the action of chlorine on alcohol. It has been introduced into medicine within a few years as a substitute for opium, as a sedative, narcotic, and hypnotic. It is generally efficient in doses of twenty to thirty grains; but much larger quantities have been used, not only with impunity, but with actual benefit. Nevertheless, its employment in large doses has occasioned dangerous and even fatal results in a number of reported instances. The fatal result has usually been sudden, the victim passing from sleep to death without any remarkable symptoms.

Considerable discrepancy exists as regards the *fatal dose* of chloral hydrate. A large number of cases has been collected, showing, on the one hand, that a fatal result not unfrequently follows an ordinary dose, and, on the other, that the most enormous quantities may be swallowed with impunity, and this too without any rejection of the drug by vomiting. Dr. B. H. Richardson, of London, considers two drachms to be the maximum safe dose for an adult: under any circumstances he regards three drachms as a *fatal dose.* Evidence, however, can be adduced to show that a much larger amount may be taken without a necessarily fatal result. Dr. Ludlow reports the case of a nurse in the Philadelphia Hospital who was believed to have swallowed *four hundred and sixty grains.* She was found in an unconscious condition, from which she was with great difficulty rescued by vigorous flagellation, and by the application of electricity. (Phila. Med. Times, Oct. 15, 1870.) Dr. Williams relates (Balt. Med. Jour., Feb., 1871) the case of a man who took, it was supposed with suicidal intent, *about six hundred grains* of chloral, the only appreciable effect of which was the production of profound coma, lasting eighteen hours, and ending in complete recovery without any medical treatment. The purity of the drug was shown by an examination of the portion left in the package. The above is probably the largest dose on record not attended with fatal consequences.

On the other hand, Dr. H. W. Fuller reports a case in which thirty grains produced alarming symptoms; and another, in which the same dose caused death (Lancet, March 27, 1871). Dr. N. R. Smith, of Baltimore, met with cases in which sudden death followed ordinary doses; and one instance in which an enema containing ninety grains produced rapid insensibility, and death in three hours (Lancet, 1870, ii. p. 476). From what is known of this drug, it does not appear safe to administer it in doses exceeding thirty grains, or to repeat it oftener than every six or eight hours. Many instances of its fatal operation have been traced to the continuance of its use in gradually increasing doses.

The *post-mortem appearances* that have been observed comprise congestion of the brain and its membranes, with exudations in the pia mater of a sero-gelatinous character. In one case reported, the brain itself was pale and friable, with injection only of the choroid plexus.

Chemical properties.—Hydrate of chloral is a white, brittle, crystalline solid, of a peculiar odor and a pungent, bitter taste. It is not inflammable. Heated on platinum it volatilizes. It is soluble in water. Potassa added to its boiling aqueous solution converts it instantly into chloroform, which escapes with effervescence, and into formic acid, which combines with the alkali. It decomposes a salt of copper like grape-sugar.

Dr. Liebreich, of Berlin, supposes that chloral, while circulating in the blood, undergoes decomposition into chloroform and formic acid, through the agency of the alkalies of the blood. Following out this idea, its physiological and its therapeutical action have been by some ascribed to the chloroform thus produced.

Detection in organic mixtures, or in the contents of the stomach.—The principle involved in the chemical analysis has reference to the conversion of chloral into chloroform through the agency of an alkali, as explained above. The solid matters should be properly divided, water added, if necessary, and the whole rendered alkaline by caustic potassa, and heated in a flask, after the manner described under the head of CHLOROFORM.

CHAPTER XXIV.

ORDER II.—SPINAL NEUROTICS, OR TETANICS.

POISONING BY NUX VOMICA.—STRYCHNIA.

NUX VOMICA is by far the most important poison included under the Order of *Tetanics*, or such substances as primarily affect the spinal cord and excite tetanic convulsions.

It is the seed of the *Strychnos nux vomica*, a tree growing in India. Several seeds are inclosed in a yellow fruit, which is about the size of an ordinary orange. These seeds are circular disks, an inch or less in diameter, concave on one surface, and convex on the other. Their color externally is light brown; they are covered with whitish, silky hairs, radiating from the centre. The body of the seed is nearly white; its texture is extremely hard and horny, which renders it very difficult to pulverize. The interior of the seed is dyed a rich orange-color when touched with a drop of nitric acid, and is tinged green by perchloride of iron. The taste is intensely and persistently bitter. It contains two powerfully-active alkaloidal principles—*strychnia* and *brucia*, united with *igasuric* or *strychnic acid.* The quantity of strychnia has been variously estimated to amount to from one-half to one per cent. of the seed.

Nux vomica is officinal in the different Pharmacopœias in the forms of the *powder*, *extract*, and *tincture.* In overdoses it acts as a terrific poison. The symptoms, post-mortem appearances, treatment, etc., will be fully noticed under the succeeding paragraph on STRYCHNIA. The smallest *fatal dose* of nux vomica recorded is *thirty grains* of the powder (about the weight of one seed), and *three grains* of the alcoholic extract.

STRYCHNIA.—This alkaloid exists in several species of the Strychnos, besides the S. nux vomica: it is the poisonous principle of *Strychnos Ignatia*, or *St. Ignatius' bean;* and it is also found in *false Angustura bark*, which is the bark of S. nux vomica. Strychnia is often employed for the destruction of wild and other animals. It has frequently been the cause of accidental and suicidal poisoning, and of late years its use for homicidal poisoning has been decidedly on the increase. The celebrated *Palmer case*, which occurred in England in 1856, has brought it very prominently before toxicologists.

This alkaloid is found in the shops both as a white powder and in colorless crystals. It has several distinct crystalline forms: those most commonly seen are the rectangular prism, and the octahedron. If allowed to crystallize from a drop of the solution of the acetate, by exposure to the vapor of ammonia, it is exhibited usually in the form of well-marked, lengthened prisms, which cross one another at an angle of 60°. If allowed to crystallize from an ethereal or chloroform solution on a glass slide, a variety of forms will be noticed, such as prisms, rosettes, plumose leaves, crosslets, and stellate needles. The fact of the diversity of the crystalline forms of strychnia should be borne in mind by the toxicologist, lest an undue importance be given to this branch of the investigation in a criminal case. The special chemical characters of strychnia will be described hereafter.

Symptoms.—The symptoms occasioned by nux vomica and strychnia are similar in kind, though they vary somewhat in the rapidity of their development, which is doubtless owing to a difference in the rapidity of their absorption. As a general rule, the symptoms appear soonest after taking strychnia. In either case, after the ingestion of a large dose, the individual first experiences a feeling of general uneasiness and restlessness, with a sense of impending suffocation, and of a want of air; very soon twitching of the muscles and jerking movements of the limbs and head come on. These are followed suddenly by a violent tetanic convulsion, which may pervade the whole body; the legs are stretched out stiffly and involuntarily, widely separated from each other, and the feet arched, and usually incurvated or turned inwards. The arms are flexed, and tightly drawn across the chest; the head is rigidly bent back, and the whole body arched backwards, so as to rest upon the head and heels (*opisthotonos*). As the muscles of the abdomen and chest are rigidly contracted, the respiratory movements become arrested; the face is congested and livid, especially about the lips; the eyes prominent and staring; the pupils dilated; the muscles about the mouth contracted to such an extent as to impart a ghastly expression to the face (*risus sardonicus*); the pulse is extremely rapid and feeble. Sometimes there is foaming at the mouth, and the froth may occasionally be tinged with blood. During all this time the intellect remains perfectly clear; the patient experiences the most intense suffering, gasping for breath, and seeking in vain for relief in asking to be turned over, or moved, or held. The jaws are not always fixed during a paroxysm; the patient may hence be able to speak, and, as great thirst is one of the prominent effects of the poison, he may ask for water, but the attempt to swallow it is very apt to intensify the spasm, as in the case of hydrophobia, or to reproduce it if there is an interval of calm.

The paroxysm may last from half a minute to two or more minutes, when complete relaxation takes place; the patient feeling exhausted, and being often bathed in perspiration. In some cases, the pupils, which were dilated during the paroxysm, contract during the intermission. After a little time, varying from a few minutes to half an hour, the fit returns: it is usually preceded by a sense of the impending danger, the special senses being exceedingly acute. The spasm may be brought on by the slightest cause, such as an attempt to move, a current of air from fanning, a sudden noise, a mere gentle touching of the patient. Often it comes on without any apparent cause. In some instances the violence of the tetanic seizure has been such as to jerk the patient off the bed. In a case likely to prove fatal, the paroxysms increase both in frequency and violence, until at last death ensues, either from asphyxia, the patient dying in a paroxysm, or from apnœa, death occurring during the intermission, from pure exhaustion.

As already stated, the intellect remains perfectly clear throughout the attack, except, in some instances, just before death, where the brain may have become clouded from the effects of the asphyxia. The patient usually has a vivid apprehension of dissolution, as likewise of suffering, and while anticipating the approach of a paroxysm he will frequently ask to be held. As a general rule, when the paroxysms of strychnia-poisoning are once established, they progress either towards a fatal termination or a cure, within two hours of the seizure. Of course there are exceptions to this rule, which will be alluded to hereafter.

The *time of the first manifestation of the symptoms* varies from a few minutes to several hours: the average may perhaps be stated to be *from fifteen minutes to half an hour*. A number of cases are recorded illustrating both extremes. Thus, Dr. G. H. Barker reports (Amer. Jour. Med. Sci., Oct., 1864, p. 399) the case of a young healthy married woman, to whom had been administered, with criminal intent, not over six grains of strychnia: violent symptoms were present in *three minutes*, and death took place, in a convulsion, in half an hour after taking the poison. This is believed to be the most rapid case, in regard to symptoms, on record. In Dr. Warner's case, who took, it is supposed, less than half a grain, the symptoms were manifested in *five minutes*, and death occurred in about eighteen minutes. In a case mentioned in the Annales d'Hygiène, 1861, i. p. 133, convulsions came on in *five* minutes. On the other hand, this interval may be protracted for two or three hours. Dr. T. Anderson reports (Amer. Jour. Med. Sci., April, 1848, p. 562) the case of a gentleman who took in mistake three grains and a half of strychnia, and experienced no particular symptoms for *two hours and a half*, when he suddenly fell backwards; but, on being immediately raised, he was able to walk home. He soon felt better, and in five hours after taking the dose he repeated it. In ten minutes afterwards, he was seized with violent tetanic spasms, which continued, with intermissions, for several hours, after which he finally recovered. Undoubtedly, the *form* in which the poison is administered will have considerable influence on the rapidity with which the symptoms will be developed. This is shown in a case cited by Dr. Taylor (Prin. and Prac. of Med. Jurisp., 1873, p. 405), that of a boy, aged twelve years, who swallowed a pill containing three grains of strychnia, in whom no symptoms were shown for *three hours;* they then set in in the usual way, and death took place in ten minutes. The pill had been prepared with mucilage, eight months before, and was consequently hard and difficult to dissolve. In the celebrated Palmer case, Cook took two pills containing strychnia: no symptoms were observed for *an hour and a quarter*, after which death occurred in twenty minutes. It is unnecessary further to multiply examples of this diversity in the length of time before the exhibition of the symptoms of strychnia-poisoning: although the delay may often be ascribed to the form in which the poison is administered, there are cases in which the unusual delay cannot thus be accounted for, but where it must be referred to some individual peculiarity of the patient.

Dr. Wormley (Micro-Chem. of Poisons, p. 40) mentions a case (recorded in the Chicago Med. Jour., Nov., 1860) where the remarkable postponement of the appearance of the usual symptoms seemed to be owing to the effects of large doses of opium taken simultaneously. Three grains of strychnia, a drachm of opium, and an indefinite quantity of quinine were taken at the same time. No symptoms of any kind appear to have been observed for *twelve hours;* and then they partook of a mixed character, slight tremblings alternating with cerebral symptoms. Another remarkable case is reported in the "American Journal of the Medical Sciences," January, 1863, p. 259. A young druggist, with suicidal intent, at half-past eight o'clock at night, swallowed between eight and ten grains of strychnia in an ounce of bitter almond water. A little later, he took an additional dose of twelve grains of strychnia. Feeling nothing peculiar, he took, at nine o'clock, ten grains of acetate of morphia dissolved in an ounce of bitter almond water, and then lay down in bed. Ten minutes later, to hasten his death, he poured chloroform on his pillow. Partial insensibility now manifested itself, and continued for about an hour and a half, when he was seized with violent cramps and cessation of respiration, but without pain. Loss of consciousness then supervened, but he soon revived and had another attack of convulsions. Emetics and tannic acid were now administered, and two days afterwards no trace of poisoning remained. In both the above instances we must suppose that the ordinary impressions of strychnia were undoubtedly modified and, so to speak, held in abeyance by the powerful doses of the narcotic taken along with it. Nevertheless, in some experiments of our own on animals, with strychnia and morphia combined, the latter poison, far from antagonizing the former, appeared rather to intensify it (see *ante*, p. 96).

If the poison be injected subcutaneously, or even applied to the healthy mucous tissue, its effects are manifested much more speedily. Dr. Shuler relates a case of amaurosis, in which about one-twelfth of a grain of strychnia was introduced into the corner of the eye. In three or four minutes symptoms of poisoning appeared, such as convulsive respiration, violent tetanic shocks, and an appearance of impending death. The patient, however, ultimately recovered. (Med. Times and Gazette, July, 1861.)

Some interesting clinical experiments of Prof. J. J. Chisolm, of Baltimore, with strychnia on patients affected with amaurosis, show very conclusively that the human system speedily acquires a remarkable tolerance of this substance. Finding that doses of strychnia which at first were beneficial in this form of disordered vision soon ceased to be effective, Dr. Chisolm was led to augment the quantity gradually, until he reached the large amount of half a grain per diem, given in three separate doses. It was also ascertained that as regards this tolerance of the drug it makes no difference whether it is administered hypodermically or by the stomach. (Amer. Jour. Med. Sci., April, 1872, p. 342.)

Fatal dose.—The susceptibility of different individuals to the action of strychnia varies greatly, as in the case of other poisons. The average medicinal dose is one-sixteenth of a grain : it is customary to commence with a rather less quantity, and gradually increase it up to one-twelfth or one-eighth of a grain. According to Taylor, one-sixteenth of a grain has proved fatal to a child between two and three years old, in four hours. Dr. G. B. Wood mentions the case of a lady who was thrown into alarming spasms, almost threatening suffocation, by *one-twelfth of a grain* (Therapeutics, vol. i. p. 834). Wormley cites another instance reported by M. Duriau, where one-sixth of a grain, taken by a woman aged twenty-eight years, produced, ten minutes afterwards, violent tetanic convulsions, in which the whole body became rigid; these continued at intervals, and were succeeded by a sense of burning in the epigastrium and pharynx and great irritability of the stomach, which lasted for six weeks (*loc. cit.*, p. 542). The smallest fatal dose for an adult (where a record has been kept) is *half a grain*—in the case of Dr. Warner. Prof. Guy states that "a quarter of a grain may destroy life," without, however, specifying the age of the subject. Dr. Ogston reports a case where *three-quarters of a grain* killed a man in three-quarters of an hour. Instances are numerous in which death has resulted from a grain and upwards of this poison. A fatal dose of strychnia for an adult may be stated to be from half a grain to a grain.

It would appear, from some recorded instances, that this poison possesses somewhat of a cumulative power. It is, however, more probable that after the system has become, as it were, saturated with the drug, from its continued use in small doses, a very slight increase of the dose may develop alarming symptoms.

On the other hand, numerous recoveries have occurred after swallowing very large doses of strychnia, sometimes with, and sometimes without, any antidote. In the "Lancet," for 1863, i. p. 54, Dr. Angell describes the case of a girl who recovered in six or seven hours from a dose of *four grains* of strychnia. Her convulsions were very violent, with difficult respiration, and fear of impending death. She had only three paroxysms. Another instance of recovery, after taking *seven* grains, is given in the "Medical Gazette," vol. xli. p. 305. Wharton and Stillé quote three cases of recovery after *four grains* of the poison had been swallowed (Med. Jurisp., 1873, ii. p. 575). Prof. Wormley mentions an instance related by Dr. Givens, where a young man swallowed, with suicidal intent, two large pills containing not less than *ten* or *twelve grains* of strychnia. Violent convulsions set in, which, however, subsided in seven hours, but leaving the patient in a prostrate condition, from which he entirely recovered in a week. Very early vomiting had occurred in this case, to which the immunity may have been due. Dr. Wilson has reported a case (Amer. Jour. of Med. Sci., July, 1864, p. 70) where a young man, aged twenty-two years, recovered in fifteen hours, although he was believed to have taken *forty grains* of strychnia. Early vomiting also occurred in this case. In these instances, where excessive doses have been taken, there is strong reason for believing that the poison was not of full strength, but was probably adulterated with some inert substance.

Fatal period.—This, like the fatal dose, is liable to a considerable variation. In the case of Dr. Warner, already mentioned, death occurred in about *eighteen* minutes, after taking not over half a grain of strychnia. Dr. Taylor records two cases—those of a man and wife—that occurred in Belgium, in 1870, of still more rapid death. The husband died in a *quarter of an hour* after taking seven grains and a half of what he supposed to be chloride of morphia, but which proved to be largely mixed with strychnia; and the wife, who took a similar dose, died in *ten minutes.* Dr. J. Gray refers to a case which proved fatal in *five* minutes. In Dr. Barker's case, already alluded to, six grains caused death in *thirty minutes;* and in Dr. Theinhart's case (Amer. Jour. of Med. Sci.; Jan., 1848, p. 303), thirty grains of the poison proved fatal in *half an hour.*

On the other hand, life has been prolonged, even after taking large doses, for several hours. In the case of Cook, poisoned by Palmer, death occurred in about an hour and a quarter after swallowing the poison. In a case reported to Dr. Taylor by Mr. Wilkins, death did not take place until *six hours* after taking three grains. In a case of a woman examined by the author, in 1861, death did not occur until *after six hours* after swallowing six grains of strychnia. (See report of this case in Amer. Jour. Med. Sci., Oct., 1861, p. 409.) These two last-mentioned cases are probably the longest on record, as regards the fatal period. In the majority of instances the poison destroys life *within two hours.*

In poisoning by *nux vomica*, death may occur within two hours; but a case is reported by Christison in which a man died in *fifteen minutes* after taking a dose. This is probably the shortest period known.

Treatment.—Prompt and free emesis is of the greatest importance. Copious draughts of warm mustard and water, or a mixture of sulphate of zinc and ipecac, answer well for this purpose; or the stomach-pump may be resorted to. On account of the rigidity of the jaws and the difficulty of swallowing, it may be impossible to get anything down the throat; the attempt to do so often bringing on a convulsion. We would strongly recommend the employment of *chloroform* by inhalation in all these cases. Numerous recoveries under its use attest its great value. Dr. Clark (Buffalo Med. and Surg. Jour., Nov., 1866, p. 135) reports the case of a man laboring under delirium tremens, who swallowed over twenty grains of strychnia: there was early vomiting, and the patient was kept under the constant use of chloroform for eight consecutive hours, during which time all attempts to suspend its use were attended by a recurrence of the symptoms. In eighteen hours he was convalescent. Dr. Dresbach, of Ohio, who appears to have been the first to administer chloroform in poisoning by strychnia, gave two drachms internally to a man who, by mistake, had swallowed a solution containing three grains of the poison, and who had most violent symptoms in twenty minutes. There was complete relief in less than twenty minutes afterwards. (Am. Jour. Med. Sci., April, 1850, p. 546.) A case is also mentioned in the "United States Dispensatory," 1865, p. 1357, where a young man took four grains of strychnia, and had most violent tetanic spasms. Complete recovery took place under the use of chloroform, administered by inhalation and by the stomach. The patient was kept under its influence for thirteen consecutive hours, during which time two pounds of the anæsthetic were consumed by inhalation.

Another physiological antidote is *bromide of potassium.* From the known efficacy of this drug in controlling ordinary convulsions, it might be inferred that it would exert some power over strychnia-convulsions. Many instances of its influence over the latter might be cited. Dr. C. B. Gillespie, of Freeport, Pa., reports the case of a man who, after swallowing fully two grains and a half of strychnia, in about two hours was seized with the usual tetanic spasms and other symptoms pertaining to this poison. After administering a teaspoonful of tincture of hyoscyamus (the only medicine at hand), eighty grains of bromide of potassium, dissolved in half an ounce of water, were given to him every half-hour. The paroxysms gradually became less violent and frequent, and had entirely ceased by the time the last dose was taken. In thirty-six hours he had completely recovered. (Amer. Jour. Med. Sci., Oct., 1870, p. 420.) Another instance in which the bromide of potassium seemed to act antidotally is related by Dr. W. W. Hewlett, of Babylon, L.I. (N. Y. Med. Jour., March, 1871). A farmer, aged thirty years, took, by mistake, five grains of strychnia at night, immediately after which he went to bed, and slept for two hours: he then awoke "feeling very much confused." Pains in the abdomen, with twitchings of the limbs, ensued, which were soon followed by violent tetanic convulsions. As he was of intemperate habits, and had been accustomed to the use of elixir of opium, his friends gave him this medicine, supposing his spasms were owing to drink. He took six teaspoonfuls of it in the course of two hours. Nausea and vomiting soon ensued, after which he felt better, and remained quiet for two hours. The convulsions now returned, and so continued alternately, with vomiting artificially excited, until five o'clock next morning. At this time the convulsions had returned with great violence. As a *dernier ressort*, bromide of potassium was ordered, in doses of ninety grains and upwards, every half-hour. In twenty minutes after taking the first dose, the improvement was perceptible. The bromide was then given in drachm doses, every hour; but the convulsions coming on again with greater severity, the remedy was given every fifteen minutes, for one hour. Again he felt better. The bromide was now administered in smaller doses for a day and a half. In thirty-six hours after taking the poison he was able to walk about, feeling a little weak, and occasionally a slight twitch.

A few cases have been put on record in which *hydrate of chloral* has been successfully used in the treatment of strychnia-poisoning. In the "Glasgow Medical Journal" for February, 1871, Dr. J. St. Clair Gray gives the results of numerous experiments made with various alleged antidotes for strychnia, on the lower animals. After alluding to chloroform, Calabar bean, woorara, and chloral, as the substances most likely to neutralize the effects of strychnia, though admitting their inefficacy if administered after the accession of the tetanic spasms, he suggests the trial of *nitrite of amyl* as being well worthy of confidence in a case of strychnia-poisoning in man. In his hands it proved more efficacious (although by no means always successful) than any of the other reputed antidotes.

The latest proposed antidote is *atropia*, which appears to exercise a true antagonizing effect over strychnia. Mr. S. Buckley relates (Edin. Med. Jour., Sept., 1873) an interesting case of this in a woman, aged twenty-eight, who had taken, at 4 P.M., an unknown quantity of strychnia. When brought to the Manchester infirmary, half an hour afterwards, she was in a state of perfect opisthotonos; the spasms painful and severe, and the intervals short. After chloroform had been administered ineffectually, atropia was given in repeated small doses, with remarkable benefit. The patient ultimately recovered. (See *post*, ATROPIA, for fuller details of this case.)

Among other reputed antidotes, *tobacco* has been recommended. This substance, originally proposed by Prof. Haughton, of Oxford, on physiological principles, has been fully tried, by various authorities, but without encouraging results. For the purpose of testing its antidotal powers, Prof. Wormley made thirteen experiments upon cats, administering to each animal half a grain of strychnia, along with an infusion of twenty grains of tobacco, and in some instances repeating the latter substance. As the result of these experiments, all the animals, with a single exception, died, and, in most instances, within the usual period. In some of the cases the strychnia-symptoms did not appear to be at all modified by the tobacco; while in others they were of a complex character, indicating the action of both the toxic agents. In the exceptional case mentioned, the animal that had taken the mixed poisons, after vomiting, and exhibiting some stiffness of gait, appeared to have completely recovered in the course of an hour. These negative results with tobacco are entirely confirmed by our own experiments made on dogs, and reported in the "American Journal of the Medical Sciences," April, 1871, p. 382, *et seq.* In some of these there did not seem to be the slightest antagonizing influence exerted by it; and in one only did the toxic impression of strychnia appear to be at all modified by the tobacco. In the latter instance, although some controlling influence was undoubtedly exerted, it could hardly be said to amount to an *antagonism*, since the final fatal symptoms were those of strychnia. As regards the antidotal powers of *tincture of chloride of iron*, *tincture of iodine*, and *aconitia*, with all of which we have experimented on dogs, we have not found them to yield any results that would entitle them to confidence. (See paper above referred to, p. 385.)

Finally, in relation to the alleged powers of *tannic acid*, *animal charcoal*, *iodo-iodide of potassium*, *camphor*, etc., no reliance whatever should be placed upon them as antidotes. Doubtless, recoveries have occurred both in man and in animals, from poisonous doses of strychnia, after taking the above-named substances; but certainly in no authentic instance without early and free vomiting of the poison.

Post-mortem signs.—The characters furnished by the autopsy are not always similar, nor are they by any means characteristic. Nevertheless, as remarked by Tardieu, although there may be no positive signs by which to identify the case, certain negative signs may be deduced that are not without value, when these are compared with the symptoms exhibited during life. Probably the lesions most commonly observed are congestion of the brain and its membranes, and sometimes of the upper part of the spinal cord, with engorgement of the lungs, and a dark and fluid condition of the blood. The heart is sometimes empty and contracted; at other times it is partially empty and flaccid. The urinary bladder is empty. Congestion of the liver, spleen, and kidneys has also been observed. As usually viewed after death, the body is in an exceedingly rigid state. Although the individual may have died in a frightful tetanic convulsion, there is a universal muscular relaxation, *immediately* after death; this, at least, is the general rule (it *uniformly* occurs in animals poisoned by strychnia); but very soon—in animals as well as in man—cadaveric rigidity sets in, and is very persistent. We have known it to be exhibited in a marked degree six weeks after death. Along with the rigidity of the body, there is usually noticed a livid appearance about the mouth and tongue, also of the fingers and toes; both the latter are tightly flexed. As regards the *alimentary canal*, nothing whatever of a distinctive character is shown. It should be remembered by those who might be led to attach an undue weight to the congested state of the cerebral and spinal vessels, that precisely such lesions are seen as the result of various disorders of the cerebro-spinal cord, and which, moreover, *may have been attended with tetanic convulsions* (Abercrombie). We therefore must infer that before a diagnosis of strychnia-poisoning can be established exclusively from the *lesions*, or even from the *symptoms* conjoined (unless the latter possess all the striking characteristic marks), the absence of all disease of the spinal cord must first be unequivocally proved.

Diagnosis.—It is of the utmost importance that an expert, summoned to pronounce upon the reality of a case of alleged strychnia-poisoning, should have clear and definite ideas in relation to the symptoms presented during life, inasmuch as these, along with the moral evidences, may constitute the sole proof that can be offered in the case. We shall see hereafter that there may be an entire absence of *chemical* proof. In the celebrated Palmer case, in England, this question was most thoroughly sifted by both sides. Indeed, this very case affords an illustration of just the sort of difficulties that present themselves in forming a correct appreciation of these symptoms. The only real difficulty consists in properly discriminating between the symptoms of the poison and those of disease. In the Palmer trial, the defense brought forward a large number of diseases which, as remarked by Tardieu, "have but a faint resemblance to, and often a complete diversity from, the characteristic phenomena of strychnia-poisoning." Among those cited on the occasion mentioned were delirium tremens, eclampsia, hysteria, epilepsy, apoplexy, angina pectoris, and even syphilis! The only diseases whose symptoms could by any possibility be mistaken for those occasioned by strychnia are *tetanus*, in its varieties of traumatic, idiopathic, and hysterical, and possibly some forms of *epilepsy.* Let us briefly examine the differential signs of these disorders.

Tetanus constitutes the really important disorder whose symptoms might be possibly confounded with those of strychnia. If the expert were obliged to decide *solely from the convulsion*—apart from its mode of invasion and seizure, its duration and termination, the condition of the intervals between the paroxysms, in fine, apart from the whole history of the attack—he might be unable to discriminate between the disease and the poisoning. But where a careful examination of all these attending circumstances has been instituted, there can be no possible difficulty in reaching a satisfactory conclusion. The really distinctive characters are the following: (1) In *traumatic* tetanus, the history of the case, as being connected with some injury, such as a lacerated or contused wound, involving tendons, nerves, fasciæ, and aponeuroses, will always throw sufficient light on the case to admit of an easy diagnosis; but it must not be forgotten that a very trifling injury (apparently), such as a small splinter of wood getting beneath the fascia of a limb, or the accidental insertion of a nail into the hand or foot, may after the lapse of several days bring on this frightful disorder, while the patient in the mean time has entirely forgotten the real cause. Such a case might possibly be mistaken for *idiopathic* tetanus. As regards the latter form of this disorder (idiopathic), the first point to be noticed is its extreme rarity, especially in temperate climates; secondly, its mode of invasion (as likewise that of traumatic tetanus), the duration of the attack, and the character of the symptoms are entirely different from those of strychnia-poisoning. In idiopathic tetanus, according to Bouillaud, Valleix, and Gimelle, there are always certain prodromes, such as chills, faintiness, insomnia, vertigo, headache, and painful tension about the diaphragm, which may last several days. These, of course, are entirely wanting in poisoning by strychnia; and they never can be mistaken for the agitation and general uneasiness which precede, *for only a few minutes*, the sudden outburst of convulsions, in the case of the poison. Thirdly, the first phenomena that characterize the invasion of tetanus are the *painful stiffness of the neck and jaws*, and a difficulty of moving the head; then, after some passing convulsions over the different muscles of the body, the rigidity spreads generally to the trunk, and thence to the limbs. In some instances the contractions reach their greatest intensity in the course of a few hours; in others, some days elapse before this takes place. In contrast with this picture, a case of strychnia-poisoning presents the following points. Instead of the gradual development of the rigid spasms observed in the case of tetanus, and commencing always in the muscles of the neck and jaw (trismus), we have, in poisoning, a *sudden* tetanic seizure of all the muscles of the body simultaneously. Here, the violent spasmodic contraction of the muscles of the neck and back, which jerks back and fixes the head as in a vise, and which arches the back like a rigid bow, can scarcely be confounded with the slow and gradual progress of the convulsions of the disease. Again, while the muscles of the neck and jaw are never the first to be affected in strychnia-poisoning, but are often the very last to be tetanized, the reverse is always the case in the disease, the *trismus* being the first indication of its approach. A fourth distinction is founded on the further progress of the two cases: whilst the violent and universal spasm produced by strychnia lasts from half a minute to one or two minutes, and is then followed by complete relaxation and absolute calm, in tetanus, on the contrary, the rigidity of the affected parts is generally permanent, and if there be exacerbations, the intervals never exhibit the character of the complete intermission witnessed in the poisoning. Fifthly, the *termination* of the cases is widely different: a case of idiopathic tetanus never terminates fatally in two or three hours; but several days usually elapse before this event. On the other hand, in a case of poisoning, after three or four rapidly-recurring paroxysms, death may occur in a period varying from less than half an hour to two hours: as a rule, recovery may be expected if the patient survives the last-mentioned period.

Dr. L. Starr reports a case of traumatic tetanus fatal in less than twelve hours after the first appearance of muscular twitchings, and within one hour and a half after the first convulsion (Phil. Med. Times, vol. iii. p. 311). A still more rapid development of symptoms occurred in the remarkable case witnessed by Prof. Robinson, of Edinburgh,—that of a negro who lacerated his thumb by the accidental fracture of a china dish. He was seized with convulsions almost instantly, and died with tetanic symptoms in a quarter of an hour (Watson's Lectures, art. *Tetanus*).

There are also other differential signs, such as the mode of contraction of the arms, hands, and feet, the peculiar feeling of apprehension and alarm before each paroxysm, the cry of the patient, &c., which, although not positively diagnostic, are nevertheless highly suggestive of the presence of the poison.

As regards the *hysterical* form of tetanus, although its very existence has been denied by some, especially as connected with the male subject, other authorities of equal weight describe this variety of the disorder, and mention cases illustrative of it. It is well known that this protean affection, hysteria, may assume the appearance of nearly every other disease, and there is no doubt that in some of its phases it may put on the semblance of tetanus. Of course, in a suspected case, a knowledge of the antecedent history of the patient would serve to clear up the diagnosis, as well as to distinguish it from poisoning by strychnia.

In relation to *epilepsy*, the only other disease that need be noticed, the diagnostic signs are sufficiently distinct. The whole features of an epileptic seizure are very different from those of a paroxysm induced by strychnia. The unconsciousness alone would serve to distinguish them. Then the peculiar clonic convulsive movements of epilepsy are totally distinct from the characteristic tetanic spasm of strychnia-poisoning. Again, after the termination of an epileptic convulsion there is always a deep stupor, the patient sleeping profoundly sometimes for hours; whereas the tetanic spasm occasioned by strychnia is followed by complete relaxation and wakefulness; there is no loss of consciousness whatever. Again, whilst death may possibly occur in a first epileptic fit, it has never been known to occur in the first tetanic spasm from strychnia. Nevertheless, the case is supposable where an epileptic paroxysm has terminated fatally under suspicious circumstances; here the death might, very naturally, be attributed to poison, and nothing but an accurate autopsy would be able to reveal its real cause. M. Tardieu (*loc. cit.*, p. 934) gives an instructive instance of this character, which fell under his own observation. A rich foreigner, who had long been subject to convulsive (epileptic) attacks, resulting from habits of intoxication, on one occasion succumbed to an attack, after having taken a powder of unknown composition. The disturbed interests and excited feelings of the survivors suggested suspicions of poisoning, which the position, title, and large fortune of the deceased, no less than the peculiar circumstances of his death, would not suffer to remain quiet. An echo of Palmer's crime had been sounded, and the name of *strychnia* had been pronounced. Tardieu conducted the examination of the body. He found in the brain marks of deep and old structural changes, which, in connection with the well-attested antecedents of the deceased, were sufficient to do away with all ideas of poisoning by strychnia.

Chemical analysis.—As already stated, strychnia occurs both as a white powder and in the crystalline form (see p. 397). It is almost insoluble in water; one part of the alkaloid requiring from seven to eight thousand parts of cold water for its solution. It is rather more soluble in hot water. Absolute alcohol dissolves one part in a little over two hundred; common whisky, one part in four hundred (Wormley); *amylic alcohol*, one part in one hundred and twenty-two; *benzole*, one part in two hundred and fifty; *commercial ether*, one part in about one thousand; *pure ether*, one part in about fourteen hundred; *chloroform*, one part in eight of the menstruum. From the above, it will be seen that chloroform is better adapted for separating strychnia from its aqueous solutions, than ether; although the latter solvent will answer very well, and is much employed for this purpose. The alkaloid is insoluble in the fixed alkalies, and only sparingly soluble in ammonia.

The *salts* of strychnia are for the most part very soluble in water and in alcohol; much more so in the latter fluid than the pure alkaloid. They are also very slightly soluble in ether.

The *taste* of strychnia is extremely and persistently bitter. This taste is one of its characteristic qualities. It is, in fact, the bitterest substance known. As the result of numerous experiments, we have found a distinct bitterness yielded by a solution of one grain of strychnia in several gallons of water. The books usually state the extent of this bitterness to be one grain in a gallon, or seventy thousand grains; but according to our experience it far exceeds this limit. The bitter taste we regard as one of the strongest *corroborative* proofs of the presence of strychnia, in a medico-legal investigation. Unless the ultimate extract obtained by the manipulation affords some evidence of bitterness to the taste, we need hardly expect to prove the presence of the poison by the usual chemical tests. But, on the other hand, the *mere presence* of bitterness is not evidence of strychnia, since this quality also belongs to a great number of other substances, such as quinia, morphia, aloes, colocynth, quassia, picrotoxia, etc.

The concentrated mineral acids produce no coloration with strychnia, if the latter is pure; if it contains any brucia, it will impart a reddish color to nitric acid. Heated on porcelain, it melts slowly into a brown liquid, and is decomposed, giving off dense white fumes, and leaving a carbon. If heated in the spirit-lamp, it takes fire, burning with a yellowish, smoky flame. According to some authorities, it cannot be sublimed without undergoing decomposition. Prof. Guy, on the contrary, asserts that the *sublimation test* is an admirable mode of identifying it, even with such minute quantities as the one-hundredth down to one ten-thousandth of a grain. The mode of procedure is to place the minute fragment on a clean, dry porcelain lid, in the centre of a ring of glass; a glass disk or microscope-slide is dried, heated in the flame of a spirit-lamp, and placed on the ring. The flame is then applied to the porcelain until it is sufficiently heated, when a mist will appear on the glass; and upon this, in succession, several milk-white spots, sometimes distinct and sometimes coalescing, will form. These may, or may not, be crystalline, though they often, under the microscope, present a variety of crystalline forms, some of which are pennate, or feathery. Although none of these forms, thus procured, can be considered *positively characteristic* of strychnia, they serve admirably for the further corroborative proof by the color test, which will be explained below.

1. *The color test.*—This test is so named from the beautiful succession or play of colors that is developed by it. It consists in the application of a drop of pure concentrated sulphuric acid to a small fragment of strychnia, placed on a white porcelain surface, or on a watch-glass over white paper. If the strychnia be perfectly pure, it will dissolve in the acid without coloration. If now a minute fragment of *bichromate of potassa*, *ferricyanide of potassium*, *permanganate of potassa*, *binoxide of manganese*, or *peroxide* (puce oxide) *of lead*, be stirred in contact with the solution, by means of a pointed glass rod, this play of colors is instantly manifested. At first it is a rich deep blue; this gradually passes into a violet or purple, which in its turn fades into a pink, and finally into a red. The relative duration of these different shades of color depends on the quantity of strychnia operated on, and also on the relative amounts of the acid and the color-developing substance. The success of the experiment greatly depends upon the proper proportioning of these different agents. Thus, if the quantity of strychnia be extremely small, the blue color may continue only for a moment, or it may even be entirely absent, the mixture developing only a violet or purple hue, which quickly passes into red. Where the quantity of the poison tested is almost infinitesimal, as, for example, one half-millionth of a grain, or even less, the most that can be expected, under the very nicest adjustment, as the mixture is made with the glass rod, is an evanescent flash of violet, succeeded by a reddish hue.

The *principle* involved in the color test is the action of nascent oxygen (developed by the acid on the various oxidizing substances above named) upon the strychnia. For the performance of the experiment, it is immaterial which of the color-developing substances is used, provided no impurity be present. Different authorities evince a preference for one or another, according to their individual tastes. The successful result depends much more on the *purity* of the reagents, and on the freedom of the strychnia from foreign matters. It is, however, probably true that, if the strychnia be associated with organic impurities (as it is apt to be in a medico-legal examination), the permanganate will be less likely to be interfered with than the others. As a general rule, the *pure* bichromate of potassa, used in the form of a minute crystal, answers every purpose. It has also been claimed that the primary blue tint is more intense and more durable, when developed by the permanganate, the binoxide of manganese, or the oxide of lead, than by the bichromate; but, as remarked by Dr. Wormley, this is true only of given quantities of the alkaloid, and depends upon the physical state of these different reagents.

In an excellent practical paper, by Dr. J. B. Lyman, in the "New York Medical Gazette," March 18, 1871, the subject of testing for strychnia, both in its pure state and as associated with organic matters, is fully discussed. The author thinks that for the detection of *pure* strychnia any of the usual color-producing substances may be indifferently employed; but where the poison is mixed with organic impurities, he found the most reliable reagents to be the *permanganate of potassa* and the *hydrated peroxide of manganese* (the latter is not the commercial black oxide, but is prepared by precipitating a solution of the chloride by a filtered solution of chloride of lime); the former will give the appropriate color-reaction under circumstances when the others entirely fail or are obscured. Dr. Lyman experimented with strychnia mixed with a great variety of organic substances, such as quinia, morphia, sugar, flour, tannin, etc., and also with animal matters: he found that while the permanganate would give with them, and indeed with most organic substances, *in the absence* of strychnia, a violet color, which fades away *without passing into red*, the presence of the minutest portion of strychnia always insures the true characteristic reaction, viz., the production of the *succession* of the colors—*violet, passing into pink and red successively.*

It is very important to have clear ideas about this color test for strychnia. It should be remembered that it is not the mere production of a blue or violet color by the reagents that is the diagnostic sign: other substances besides strychnia may do this; it is the *regular succession of tints*, commencing with the blue (sometimes with violet) and proceeding on to pink and red—the last continuing for a considerable length of time, and giving place ultimately to a greenish hue. So far as is known at present, strychnia is the *only* substance that responds to the above requisition. Certain substances that react somewhat similarly will be noticed hereafter under the head of *Fallacies.*

The exceeding *delicacy* of the color test deserves to be considered. If the strychnia be perfectly pure, and the manipulation be properly performed, so minute a quantity as *one-millionth of a grain* may be detected by it. This we have frequently verified in our own experience, and it is corroborated by other experimenters. The ability to identify such an almost infinitesimal quantity depends simply on the delicacy of the manipulation and the purity of the materials used. In order to procure for experimenting so minute a quantity of the poison as that above mentioned, a pure solution of strychnia in water acidulated with a little acetic acid is first made, of definite strength. This may then readily be reduced to any degree by the addition of distilled water. Fractional portions of the last solution may then be obtained by using a pipette drawn out to a capillary point, which will deposit minute droplets on a warmed, clean porcelain or glass surface. The object here is to concentrate the quantity to be experimented on into as small a space as possible. The drop should then be evaporated to dryness spontaneously; a small drop of pure concentrated sulphuric acid is then applied to the deposit by means of a finely-pointed glass rod; and, last of all, a very minute fragment of crystallized bichromate or ferricyanide of potassium, or of any of the other oxidizing agents, is placed alongside of the acid solution, and then by means of the pointed rod it is drawn through the solution, and gently stirred with it. Dr. Lyman advises (*loc. cit.*), in experimenting with these very minute amounts of strychnia, to employ an extremely small drop of sulphuric acid, and to dilute the powdered bichromate with some inert powder (such as that scraped from a slate-pencil), and to use but a minute portion of the latter. He recommends to rub up the permanganate of potassa or the hydrated peroxide of manganese with a drop or two of sulphuric acid on a porcelain surface, with a glass rod, and to use a small portion of this solution along with the sulphuric acid, on the dried residue. For this fractional testing he rather gives the preference to the permanganate and the ferricyanide of potassium, as the oxidizing agents. Dr. Taylor regards the permanganate as objectionable, on account of its solubility, and of its being already colored; but if it is used as above directed, the objection does not seem to hold good.

Interferences.—The color test properly applied will detect an exceedingly small amount of *pure* strychnia, as above mentioned. There are many organic substances, however, whose presence will serve materially to modify, and even completely to mask, the usual color-reactions. Brieger, in 1850, first announced that the reaction was more or less interfered with by *morphia*, *quinia*, and *sugar* (Chem. Gaz., vol. viii. p. 408); and since then the list of such substances has been much extended. Dr. Wormley has likewise investigated this subject (Ohio Med. Jour., Jan. and March, 1864), with similar results.

Dr. Lyman also experimented on strychnia in the presence of morphia (*loc. cit.*). He first tried a solution containing twenty-five parts of morphia to one of strychnia, and this was deposited in minute fractional drops on porcelain, and dried; spots were also formed containing the same amount of morphia, but no strychnia. To both, the tests were applied in the usual way. *None of the tests except the permanganate gave any satisfactory evidence of the strychnia:* this, however, gave a purple color, *passing to light red.* The test applied to the morphia alone, also gave a light purple color, which faded gradually away, *without passing to the red.*

When strychnia is mixed with a large excess of *sulphate of quinia*, it refuses to respond to any of the color tests except the permanganate, which gives the characteristic change of colors. The latter will also give to quinia alone, as to morphia, a violet color, which, however, gradually fades away *without changing to red.* (*Ibid.*)

The *fact* of these interferences must, then, be admitted. Practically, however, they may be avoided in a medico-legal investigation, by the employment of chloroform instead of ether as the solvent to extract the strychnia from organic mixtures; the interfering substances (morphia, the nitrates, tartar emetic, etc.) are insoluble in this menstruum.

That *morphia*, among other substances, when associated with strychnia *in certain proportions*, has the property of disguising the usual color test of the latter alkaloid, is abundantly established, although the proposition has been denied by some eminent authorities. The author made an elaborate series of experiments for the purpose of settling this question, the results of which were published in the "American Journal of the Medical Sciences," October, 1861, and April, 1862. These results very clearly prove that when these two alkaloids are mixed together *in minute quantities*—either in their pure state, or associated with complex organic matters—the usual color test for strychnia is certainly interfered with; and, further, that the *degree* of interference "*depends upon the relative quantity of the two alkaloids,—the strychnia not being discoverable when the morphia is in excess, and barely discoverable when in equal quantity.*" To establish this proposition the following experiments are quoted from the above-named article:

"*Exp.* 1. The one-hundredth of a grain of strychnia was put into a pint of water, together with several ounces of beef finely cut up, some starch, common salt, and a few drops of acetic acid (the object being as nearly as possible to represent the contents of the stomach after eating). The whole was subjected to a moderate heat, strained, pressed, and evaporated, and finally treated after the process of M.

Stas. The ethereal solution on being concentrated yielded clear proof of the presence of strychnia, both by the color test and by the bitter taste.

"*Exp.* 2. This was a repetition of the last, except that to the one-hundredth of a grain of strychnia there was added three times that quantity of morphia (one-thirty-third of a grain). Although precisely the same process was employed, not the slightest trace of strychnia could be discovered by the color test.

"*Exp.* 3. This resembled the preceding experiment, except that the one-fiftieth of a grain of morphia was added to the strychnia (or double instead of treble the quantity): here likewise the color test entirely failed.

"*Exp.* 4. In this case an equal amount of morphia, or the one-hundredth of a grain, was added to the strychnia, the same method being still pursued: the result was that the faintest possible evidence of the presence of strychnia was afforded, and only after repeated trials."

These experiments were all purposely performed with very minute quantities of strychnia. It was not deemed necessary to experiment upon such large amounts as one grain, or, indeed, any portion over one-fiftieth of a grain. The detection of such comparatively large quantities presents no practical difficulties, either with or without the presence of morphia. But it is far otherwise when the toxicologist has to deal with such fractional portions as the *one-thousandth* down to *one five-hundred-thousandth of a grain*. In the latter case, as before remarked, the *interfering* influence of morphia becomes very apparent. As the results of various experiments made with minute portions of the two alkaloids *in the pure state*, *i.e.* free from all organic mixture, it was found that not only is the difficulty of detecting strychnia greatly increased by increasing the *proportion* of morphia, but also that the actual amount of it discoverable is nearly in the inverse ratio with the amount of combined morphia. For example, when the quantity of the two alkaloids was in the proportion of one to one, so small a quantity of strychnia as *one five-hundred-thousandth of a grain* could be detected. From this, the minimum amount discoverable progressively rose with the increase of the proportion of the associated morphia, until in the last experiment, made with one proportion of strychnia to *twenty* of morphia, the smallest quantity that could be detected had increased to one five-thousandth of a grain. Doubtless, if the relative quantity of the morphia had been further increased, the minimum quantity of strychnia discoverable would also have progressively augmented.

It must be borne in mind that the above-mentioned *minute* quantities have reference only to the alkaloids when employed in *the pure state.* But in order more fully to settle the question, and to meet the objection that the result might have been different if the two alkaloids had been *in the stomach of a living animal*, the following experiments were made. "Half a grain of pure strychnia was given to a cat, which died in convulsions in eleven minutes. The stomach, examined on the following day by Stas' process, afforded clear proof of the presence of strychnia by the color test. To a second cat, a quarter of a grain of strychnia and the same quantity of morphia were given; and the stomach was examined as before. The faintest possible evidence of strychnia was obtained,—exactly coinciding with a previous experiment made with the organic mixture. To a third cat, one-twentieth of a grain of strychnia and one-tenth of a grain of morphia (double the quantity) were given. Here there was a total failure to obtain the color test; although the bitterness of the extract, and the fact that the solution produced the characteristic tetanic convulsions in a number of frogs, distinctly proved its existence."

We are therefore of the opinion that the proposition laid down has been abundantly established by the foregoing experiments, viz., that "in experimenting on *very minute quantities* of strychnia, the presence of morphia has the power of disguising the usual color test, *especially if the latter alkaloid be in excess.*"

Certain authorities have taken exception to the above conclusions, and have maintained that there is no such interference. The late Prof. R. P. Thomas, of the Philadelphia College of Pharmacy, gives this as his opinion (Amer. Jour. Med. Sci., April, 1862, p. 340), based upon a number of his experiments; and the distinguished authors of the "United States Dispensatory" (twelfth edition, 1865) adopt the same view, quoting Prof. Thomas's paper as the basis of their opinion. But both these authorities have fallen into the error of not distinguishing between an experiment made with the two alkaloids strychnia and morphia, where *ether* was employed as the ultimate solvent (as in Stas' process), and one in which *chloroform* was the solvent used: in the former case, *both* alkaloids would be dissolved, whereas in the latter, *only the strychnia* would enter into solution (since morphia is nearly insoluble in chloroform). Of course, then, since Prof. Thomas used chloroform instead of ether (which latter was the solvent employed in our experiments), he did not meet the mooted question at all, for, as we have seen, he did not, in point of fact, experiment upon the *combined* alkaloids, *because there was no morphia present:* it had all been left behind in the alkaline aqueous solution!

In order that we may not be misunderstood, we quote verbatim from Prof. Thomas's paper (*loc. cit.*, p. 343): "The solution of caustic potassa was selected for several reasons: . . . it dissolves morphia, but does not dissolve strychnia." "As the solution of potassa dissolves morphia and rejects strychnia, while chloroform has the reverse property of taking up the strychnia and rejecting the morphia, it must be evident that the conjoint use of these fluids would effect *an entire separation* of the two alkaloids."

In the only experiment out of the many detailed by Prof. Thomas that even *appears* to lead to a result different from the results of the author, he says: "equal weights [the *amount* is not stated] of the pure alkaloids were rubbed together in a mortar and tested." Here, comparatively *large* quantities of the solid alkaloids were subjected to the experiment. Of course, as we have explained above, he could not fail to get the color test, even though the morphia might be "twenty times" in excess of the strychnia; but no mention is anywhere made of similar experiments performed upon *very minute portions.*

Finally, upon this branch of the subject, we entirely assent to the recommendation of Prof. Thomas—which is also that of all the best modern toxicologists—to employ chloroform instead of ether as the proper ultimate solvent for strychnia, *especially in the presence of morphia.*

In practice, the toxicologist will probably encounter the most troublesome *interference* from the complex organic substances which are so apt to be present along with the strychnia in the ultimate extract obtained either by chloroform or by ether. The proper method of effecting their separation will be described below, under the head of *Detection in Organic Mixtures.*

Fallacies.—Exceptions have been taken to the color test on the ground that other substances besides strychnia will yield colors somewhat similar, if not identical, when subjected to the same reagents. The substances alluded to are *curarine* (the active principle of woorara), *aniline*, *veratria*, *cod-liver oil*, *salicine*, *santonine*, *pyroxanthine*, *narceia*, *papaverine*, and *solania.* But in relation to all of them, with one or two exceptions, it may be remarked, as a radical ground of distinction, that they are colored by sulphuric acid *alone*—which is not the case with strychnia. A salt of *aniline* is not colored by sulphuric acid alone, but on the addition of bichromate of potassa it acquires a yellowish or greenish tint, shows bluish streaks, and finally assumes a deep-blue color, which lasts for some time, but ultimately becomes nearly black. In this case, the only resemblance is the deep-blue color; but the point of divergence is the relative sequence of the colors: in the case of strychnia, the blue always appears first, and is soon succeeded by the violet and red, and ultimately the *green;* whilst in the case of aniline, the blue is very slowly developed, but is very permanent, giving place finally to a *black* hue.

As regards *curarine*, the active principle of woorara or curara, there are some points of resemblance with strychnia which deserve notice. Like strychnia, it has an intensely bitter taste, and it yields under the combined action of sulphuric acid and an oxidizing agent, as bichromate of potassa, a succession of colors very much resembling those of strychnia. Moreover, its solution forms with the bichromate solution a yellow, amorphous precipitate, which, when touched with sulphuric acid, gives the characteristic play of colors. The points of *difference* are, that it and its compounds are uncrystallizable; it is colored by sulphuric acid *alone;* it is nearly insoluble in chloroform, and readily soluble in potash, and its solutions are not precipitated by the alkalies. Its *physiological effects* are the opposite of those of strychnia.

Cod-liver oil, when treated with sulphuric acid *alone*, yields a play of colors very similar to that produced by strychnia. In relation to the other substances mentioned, they all present characters abundantly sufficient to distinguish them from strychnia.

2. *The galvanic color test.* — This method, devised by Dr. Letheby, acts on the same principle as the ordinary color-developing substances, by the evolution of nascent oxygen, but in this instance by means of galvanism. A drop of a very dilute solution of strychnia is placed in a small platinum capsule, allowed to evaporate to dryness, and then moistened with a drop of strong sulphuric acid. The capsule is then connected with the positive pole of a single cell of a Grove's or Smee's battery, and the acid touched with the platinum terminal of the negative pole. In an instant the violet color will flash out with great intensity, and will remain on removing the pole. We do not think this method offers any peculiar advantage over the test as commonly applied. It is, moreover, quite as much open to interferences and fallacies as the former; and in our hands it has not been quite so successful for the recognition of very minute portions of the poison.

3. *Potassa and ammonia.*—These alkalies precipitate from somewhat concentrated salts of strychnia a white, amorphous deposit of the pure alkaloid, which shortly assumes the crystalline form. The best mode is to expose a drop of the strychnia solution for a few moments to the vapors of ammonia, and place it under the microscope: the formation of the mass of long stellate crystals can easily be distinguished. The true character of the crystals may readily be determined by touching them with a drop of strong sulphuric acid and a fragment of bichromate of potash, when the play of colors will take place.

4. *Bichromate of potassa.*—A solution of this salt produces with a solution of a salt of strychnia an immediate bright-yellow precipitate, which speedily becomes crystalline, and is insoluble in an excess of the precipitant. If the alkaloidal solution be dilute, the formation of the crystals is delayed: it may be facilitated by stirring the mixture with a glass rod. Seen under the microscope, this crystallization is very satisfactory: it usually consists of dendroidal groups, intermingled with small octahedral plates. These crystals must be confirmed by touching them, when dry, with a drop of pure concentrated sulphuric acid, when immediately the characteristic succession of colors will be developed. By this method one five-thousandth to one ten-thousandth of a grain of strychnia may be identified; and also by it, two of the most characteristic tests may be applied to the same portion of strychnia.

5. *Picric* or *carbazotic acid.*—This reagent is especially recommended by Prof. Guy, as one of the most reliable corroborative tests for strychnia. An aqueous (or alcoholic) solution of the strength of one two-hundred-and-fiftieth is used, a drop of which is added to a drop of the strychnia solution on a glass slide, and is viewed under the microscope. An abundant yellow precipitate first forms, which is soon converted into tufts of crystals of a peculiar claw-like form, resembling tufts of grass. This peculiar crystalline form seems to belong exclusively to the carbazotate of strychnia. The deposit may also be subjected to the color test, by dissolving it in a drop of sulphuric acid, and adding one of the usual oxidizing bodies.

6. *Corrosive sublimate* in solution causes an abundant white precipitate, which assumes the form of groups of radiating crystals attached to a granular nucleus. This test, according to Wormley, fails in solutions weaker than one five-hundredth of a grain of strychnia to the drop of water. It is consequently much less delicate than the preceding tests.

7. *Ferricyanide of potassium* produces in *strong* neutral solutions of strychnia a yellowish, amorphous precipitate, which is soon converted into a mass of beautiful groups of crystals. This test is likely to fail in solutions of less strength than

one grain to five hundred of water. The deposit may (as in the bichromate of potassa test) be subjected to the color test simply by the addition of a drop of sulphuric acid, which will develop the usual play of colors. In point of delicacy, however, it is much inferior to the bichromate test.

8. *Bichloride of platinum* occasions in solutions of salts of strychnia a pale-yellow, amorphous precipitate, which soon assumes a crystalline form, which is insoluble in acetic and dilute nitric acids. It will give a perceptible reaction when the strychnia is diluted to one ten-thousandth. This reagent also yields precipitates with many of the other alkaloids, of a similar color; but the crystalline forms are different in the other instances.

9. *Iodated iodide of potassium.*—The aqueous solution of iodine in iodide of potassium will precipitate a number of organic substances, and particularly the alkaloids. In a strychnia solution, even when dilute, it occasions a reddish-brown, amorphous deposit, which is soluble in alcohol, but only sparingly so in acetic acid. This deposit, after a time, assumes a crystalline form, which in the case of strychnia is peculiar, but it is readily interfered with by the presence of foreign matter. According to Wormley, from one fifty-thousandth to even one hundred-thousandth of a grain will respond to this reaction.

The precipitate thus obtained is regarded by Tardieu (*loc. cit.*, p. 953) as the best material for procuring the strychnia in a state of purity. After repeatedly washing it in water slightly acidulated with sulphuric acid, the water is decanted off, and a few drops of water acidulated with one-tenth of its weight of sulphuric acid are added, together with a pinch of clean iron-filings. In a few minutes all the precipitate is dissolved, and the liquid becomes almost colorless. When all disengagement of hydrogen ceases, ammonia, in slight excess, is added: this precipitates both the strychnia and the oxide of iron. The mass is next put upon a filter, and thoroughly washed, in order to remove the soluble salts; the filter is next carefully dried between bibulous paper, and then cut into pieces, and introduced into a flask along with pure alcohol; it is kept at a boiling temperature for an hour, frequently stirring. It is next filtered, and the filtrate evaporated to dryness. The deposit, although colored, is generally crystalline, and may be used for experiments.

10. *Sulphocyanide of potassium* yields in a moderately strong solution of strychnia a deposit which soon changes to beautiful masses of long, radiating prisms. These crystals are about the largest and best defined of the microscopic deposits obtained from strychnia.

Tardieu considers *chlorine gas* to be a very delicate test for strychnia. When a small stream of this gas is slowly passed into a small quantity of a dilute solution of strychnia, each bubble of the gas becomes surrounded by a white film, and ultimately quite a copious white amorphous deposit takes place. This is soluble in ammonia. According to the above authority, no other alkaloid gives this reaction with chlorine. Besides the above tests, there are others mentioned in the books, as *chloride of gold*, *tannic acid*, *bromine in bromide of potassium*, *iodo-hydrargyrate of potassium*, etc.

The physiological or frog test.—The extreme susceptibility of frogs to the influence of strychnia was employed by Dr. Marshall Hall as a test for this powerful poison. His plan was to immerse the frog partially in the solution of strychnia, when, sooner or later, according to the strength of the solution, the animal was seized with tetanic spasms, in which the extremities became perfectly rigid and extended. Dr. Hall states that by this method he could detect one five-thousandth part of a grain of strychnia. Dr. Harley subsequently applied the test by injecting it into the thoracic or abdominal cavity of the frog: he found that by this means one sixteen-thousandth of a grain would occasion the characteristic convulsions.

We have ourselves employed this test very extensively. In the journal already alluded to, some of these results were communicated. Undoubtedly, the frog test is one of the most delicate, as also one of the most reliable, of all the tests for strychnia, as will be seen from some of our experiments, which are here transcribed:

"A small frog, weighing about forty grains, was immersed in a solution containing one grain of strychnia to twelve pints of water (one minim of which would contain less than one ninety-two-thousandth of a grain): tetanic spasms were produced in fifteen minutes.

"A solution of one-half the strength of the foregoing, in which one minim would represent a little over one two-hundred-thousandth of a grain, produced decided convulsions in a frog weighing twenty-nine grains, after half an hour's immersion.

"The one five-hundredth of a grain of strychnia was put into the throat of a middling-sized frog: it was convulsed, and died in about thirty minutes. The extract obtained from the abdominal viscera by Stas' process, although it afforded no perceptible color test, had a bitterish taste, and produced tetanic spasms in several small active frogs.

"In these experiments, the frog was put into a small quantity of the solution—not more than about half a fluidrachm—so as simply to cover the hind legs and a portion of the body. The quantity actually absorbed through the skin must, necessarily, have been extremely minute: hence the great delicacy of the test, and its value, as a corroborative proof, in medico-legal investigations."

Prof. Wormley's experiments with the frog test gave results of similar delicacy. He found one ten-thousandth to one fifteen-thousandth of a grain, injected into the stomach of the animal, to be about the limit for this test (*loc. cit.*, p. 577).

Being desirous of ascertaining whether the presence of morphia, which, as we have seen above, has the power of disguising the color test for strychnia *in small quantities*, offers any obstacle to the employment of the frog test, we made the following experiments:

"A frog weighing forty grains was immersed in a solution containing one grain of strychnia and eight grains of morphia to a pint and a half of water: it exhibited tetanic spasms in five minutes.

"A frog weighing one hundred grains was immersed in a solution of the strength o one grain of strychnia and twelve grains of morphia in three pints of water: it exhibited the usual tetanic spasms in fifteen minutes.

"A frog weighing thirty-five grains was immersed in a solution containing one grain of strychnia and thirty-two grains of morphia in six pints of water: it was convulsed in twenty minutes. Another animal, rather smaller, was affected in five minutes.

"A cat was poisoned by taking one-twentieth of a grain of strychnia and one-tenth of a grain of morphia (double the quantity). The stomach, on being examined by Stas' process, failed to yield the color test; but the watery solution of the extract produced the most decided tetanic convulsions in *eight* frogs, generally resulting in death."

Detection in organic mixtures.—Contents of the stomach.—Any solid matters present should be cut up into small pieces, pure water added, if necessary, and a sufficient quantity of acetic acid to give a distinct acid reaction. If the elaborate process of M. Stas is to be employed, the strongest alcohol is used, instead of water, as the first solvent. In either case, the mass should be digested at a very moderate heat—about 160° F.—for several hours (a high temperature is objectionable, as it dissolves out the starchy matters). After cooling, it is to be first strained through fine muslin, and the solid residue washed with dilute alcohol, and strongly pressed. The liquid should next be concentrated at a moderate heat to a small bulk, again strained, and filtered through paper. It is generally recommended to evaporate, next, to about dryness. Any strychnia present would now be in the residue in the form of *acetate*, mixed with more or less organic matter. This residue should be thoroughly stirred with a small quantity of water containing a drop or two of acetic acid, then filtered, and the filter washed with a small portion of water; the filtrate (concentrated, if necessary) is then transferred to a stout glass tube or flask, and an excess of solution of potassa (or soda) is added, which liberates the strychnia from the saline combination. Pure chloroform, about equaling in volume the mixture, is next added, and the whole thoroughly shaken together for about a minute, when it is set aside to settle. The chloroform will dissolve out the alkaloid, and, being of greater specific gravity, it will settle to the bottom of the tube or flask.

The best practical method of separating the chloroform solution from the supernatant liquid, according to our experience, is to transfer the whole to a stoppered funnel, or, what answers quite as well, a glass syringe of proper size, after removing the piston, and having previously contracted the nozzle to a very fine aperture by means of the flame. Before introducing the liquid, the small aperture should be temporarily stopped up by means of a splinter of wood, and about half a drachm of pure chloroform should first be poured into the syringe, so as about to fill its narrow portion. The mixture is now to be carefully poured in, and sufficient time should be allowed for the subsidence of the chloroform solution. By placing the thumb over the larger aperture of the syringe, and withdrawing the wooden plug, it will be very easy to control the flow of the contents. A few drops may be allowed to fall successively, as each one dries, upon a *warmed* porcelain surface or watch-glass, for a trial test, by means of sulphuric acid and bichromate of potassa (see p. 414). The whole of the chloroform is then permitted to flow out into one or more capsules or watch-glasses, great care being observed not to allow any of the other mixture to escape along with it. It may be proper to wash the remaining alkaline liquid with a fresh portion of chloroform, shaking them together, as before, in the tube or flask, and again separating them by means of the syringe. All the chloroform is now allowed to evaporate spontaneously to dryness. The contained strychnia, if in notable quantity, may be found in the crystalline state in the deposit.

It is, however, much more apt to be in an amorphous condition when obtained from complex organic substances, as the contents of a stomach. A portion of this extract should now be examined by the taste, by the color test, and by introducing some of it beneath the skin of a small frog. The remaining portion of the extract should be dissolved in a minute quantity of distilled water, containing a trace of acetic acid. The solution, if turbid, should be filtered; and the clear filtrate should be tested with the different reagents mentioned at p. 423.

If the chloroform extract is found to be mixed with much organic matter, as denoted by its yellowish color, a drop or two of strong sulphuric acid is to be added to the solid residue and thoroughly stirred with it by means of a glass rod, and then a few drops of water added. (The acid destroys and carbonizes all the organic matter, but only converts the strychnia into a sulphate.) After standing some time, the resulting dark liquid is filtered, caustic potassa is added in excess, then an equal bulk of chloroform, and the whole shaken together as before. The deposit from this second chloroform solution is usually sufficiently pure for all practical purposes. Or, instead of treating the solid extract with sulphuric acid, the acidulated aqueous solution obtained from it may be rendered alkaline by potassa, and again shaken with pure chloroform, which will deposit the strychnia generally in great purity.

When the organic mixture is very complex, as is apt to be the case when taken from the contents of the stomach, considerable trouble may be experienced in the separation of the chloroform from the alkaline solution. It sometimes happens that, on shaking up the latter with chloroform, a white, saponaceous mass results, resembling an emulsion, from which the chloroform refuses to separate, even after standing for some hours. In such cases Dr. Wormley advises (*loc. cit.*, p. 581) to agitate the mixture with about half its volume of pure water, and allow it to repose for several hours, if necessary, when more or less of the water will separate as a highly-colored fluid; this is decanted, and the operation repeated with fresh portions of water so long as this liquid becomes colored. After removing all the water, the mixture is to be slightly acidulated with acetic acid, transferred to a small dish, and evaporated to dryness on a water-bath; the residue is stirred with a very small quantity of pure water; the solution, after filtration, if necessary, is rendered slightly alkaline by potassa, and again agitated with pure chloroform, which will now usually separate, and may be evaporated as before mentioned.

The method of *dialysis* has been recommended by some authorities as well adapted to the separation of strychnia from complex organic liquids. The details of this process have already been given (p. 113). The concentrated solution prepared from the contents of the stomach, as before explained (p. 428), are put into a dialyser, which is floated in a vessel of water containing four or five times the bulk of the former. After twenty-four hours, the diffusate is transferred to a porcelain capsule, evaporated to dryness on a water-bath, and the residue examined, as before. We have no personal experience with this process; but some carefully-conducted experiments by Wormley, as well as by others, go to show that it is not so thorough or exhaustive as the ordinary one by chloroform, before explained.

Detection in the tissues and blood.—There is no longer any doubt that strychnia is absorbed into the circulation, and deposited in the organs, just like arsenic or antimony, and that the absorbed poison may be detected in the solids of the body, and likewise in the blood. The *rapidity* with which it is absorbed is shown in a case mentioned by Taylor (Prin. and Prac. of Med. Jurisp., 1873, p. 414), where a man took five grains of strychnia by mistake, and died in half an hour. The analyst discovered in the stomach a quantity of the poison, estimated at one grain ; it was also detected in small quantities in the liver and in the tongue. This case shows that within *half an hour* four-fifths of the poison had been removed from the stomach (or at least could not be detected there by chemical means), and that in that period it had been diffused and distributed through the body. It would appear, however, that there is much variation in this respect. Thus, Prof. Casper reports a case of poisoning from five or six grains of strychnia. The deceased lived three hours and a half, and on analysis after death *three grains* of the poison were extracted from the stomach; but none was detected either in the tissues or blood (Horn's Vierteljahr. für Gericht. Med., Jul., 1864, p. 7). In this respect, strychnia is simply on a par with the other poisons, in which, as we have seen, there is often a failure to detect their presence in the tissues and organs of the body after death, under circumstances that might have been regarded as favorable for so doing.

The process to be pursued is the following. The organs to be examined should be cut up into small pieces, and, together with the fluid, digested with strong alcohol, acidulated with sulphuric acid, in the proportion of about eight drops of the concentrated acid to each fluidounce of the mixture. It should be kept at the temperature of about 180° F. for half an hour or an hour, and, after cooling, strained, filtered, and concentrated as before directed (p. 428). The residue is next to be *nearly* neutralized by caustic potassa, care being taken, however, to maintain a decided acid reaction, then filtered, and the filtrate evaporated on the water-bath nearly to dryness. To the cooled residue a drachm or two of strong alcohol is added and thoroughly stirred with it: this dissolves out the sulphate of strychnia, leaving the sulphate of potassa and organic matters. The alcoholic solution is now filtered, evaporated to almost dryness, the residue stirred with a little water, rendered alkaline by potassa, and finally agitated with chloroform, which deposits the alkaloid, if present, on evaporation.

Prof. Taylor recommends the use of alcohol and *acetic acid*, in these cases, for the extraction of the strychnia; and also *ammonia* as the preferable alkali. The impure residue obtained from the first chloroform or ether evaporation is to be purified by washing it with a few drops of sulphuric acid, adding water, neutralizing by ammonia, and again agitating with chloroform. By this process he detected strychnia in the liver of a person who died from this poison, although this organ was in a highly putrescent state (*loc. cit.*, p. 413).

A somewhat similar process has been employed for detecting strychnia *in the blood*. About four to six ounces of this fluid should be used for the analysis, which is rather complicated and tedious. The blood is first treated with water, alcohol, and sulphuric acid: the precise quantity of the latter varies for different specimens of blood; if the acid is not in sufficient quantity it fails to separate the strychnia, all of which will be apt to be retained by the solid albuminous matters. Enough of the acid should be used to merely separate a portion of the solid matters. By adopting this method, Dr. Wormley (*loc. cit.*, p. 589) succeeded in recovering strychnia from the blood of six cats and of two dogs, poisoned by comparatively small doses (as half a grain). In two of the cases, death took place in three and six minutes respectively, which shows the extreme rapidity with which this poison is absorbed.

Detection in the urine.—The urine should be acidulated with acetic acid, and evaporated on a water-bath to the consistency of syrup. When cooled, this should be stirred with about an ounce of pure alcohol, the solution filtered, the solids washed and pressed, and all the liquids evaporated to near dryness. The residue is to be stirred with a little pure water, filtered, if necessary, mixed with caustic potassa in excess, and agitated with chloroform as usual.

Failure to detect.—There can be no doubt that cases of strychnia-poisoning occur, where the most careful chemical analysis fails to detect it after death, and that, too, under apparently the most favorable circumstances. The causes that interfere with the chemical detection of poisons have already been explained (*ante*, p. 70). Of some of these failures in regard to strychnia it is difficult to give a perfectly satisfactory explanation, whilst in others the reasons may be sufficiently obvious. The mere fact of the *putrefaction* of the body is no obstacle to the chemical analysis, as has been shown in numerous instances, in some of which the poison has been recovered months after death and when the material was in quite an advanced stage of decomposition. In the celebrated case of Cook, who was poisoned by Palmer, in 1856, there was a failure to discover the poison in the stomach by Dr. Taylor; but this seems to be satisfactorily accounted for by the fact that the stomach had been tampered with before coming into his hands: it had actually been cut open and the contents lost! The above authority mentions the case of Mrs. Salter, who died from taking strychnia in two or three hours, but the most careful examination of the stomach and liver by Mr. Horsley, of Cheltenham, led to a negative result. The failure here was ascribed to the rapid absorption and elimination of the poison before death. In 1861, the author examined the body of a woman poisoned, it was alleged, by about six grains of strychnia: she survived the dose for the rather unusual period of six hours. Six weeks after death the body was exhumed, and was found to be in a good state of preservation, and very rigid. The stomach and bowels were examined two weeks later. No trace of the poison was discovered, either by the bitter taste of the ultimate extract, or by the color test, although several careful and separate analyses were made of the stomach and a portion of the intestines. Evidently, the poison had either disappeared from these organs by elimination, or some interference had occurred to obscure the usual reactions. In order to be sure of the accuracy of the process employed, we made trial with it on one-hundredth of a grain of strychnia mingled with a pint of complex organic matters,—animal and vegetable,—and succeeded without difficulty in recovering the alkaloid (see p. 418).

BRUCIA.—*Brucia* is the other alkaloid generally found associated with strychnia in the different species of Strychnos. In the *S. nux vomica* it occurs in greater quantity in the bark (*false Angustura bark*) than in the seeds, which is the reverse in the case of strychnia.

Properties.—Brucia is found in the shops both as a white powder and in the form of colorless, prismatic crystals. It is more soluble both in water and in ether, than strychnia. In chloroform and absolute alcohol it is very freely soluble. It is insoluble in the fixed alkalies, and almost so in ammonia. It has an intensely bitter taste. It forms soluble salts with the acids. Concentrated sulphuric acid dissolves it and its salts, imparting to them a faint rose color. Bichromate of potassa and the other oxidizing bodies used for the color test of strychnia, when stirred in the sulphuric acid solution, merely impart to it a yellowish color, which soon becomes greenish: there is no appearance of the color-reactions of strychnia. Hydrochloric acid causes no change of color. Nitric acid produces, with a fragment of brucia, a deep blood-red color, more intense than with morphia; this slowly fades to yellow.

The *poisonous* properties of brucia are similar in kind to those of strychnia, but much inferior in degree. They are estimated to be from one-twelfth to one-sixth weaker. As cases of poisoning by this alkaloid have occurred, and as the symptoms resemble precisely those occasioned by strychnia, although more slowly developed, the toxicologist should be on his guard against being deceived, in making a medico-legal investigation, in the event of his not discovering *strychnia* by the use of the color test. In such a case, it will be always proper for him to employ the proper reagents for *brucia.*

Tests.—(1) The most characteristic test for brucia is *nitric acid and chloride of tin.* A fragment of this alkaloid, or the dried solid residue from the chloroform or ether solution, if touched with a drop or two of strong nitric acid, instantly assumes a deep blood-red tint, and speedily dissolves into a similarly-colored solution. If this be heated, the color changes to a yellow. If, after cooling, a drop of the solution of *protochloride of tin* be added, the mixture immediately acquires a beautiful purple color, which is discharged by an excess of the tin solution, or of nitric acid. The heating of the nitric acid solution is essential if the quantity of brucia is very small, in order to obtain the reaction of the tin compound. This is a highly satisfactory test for brucia when it is carefully performed. The deep-red color produced by nitric acid on *morphia* can hardly lead to any error, inasmuch as the addition of the tin salt to the latter causes no purple color, but at most changes it to a yellow.

(2) *Sulphuric acid and nitrate of potassa.*—This test is a modification of the former. On applying strong sulphuric acid to brucia, as already mentioned, a faint rose color is produced, which, on the addition of a small crystal of nitre, changes to a deep orange-red. If the quantity of brucia be very minute —as one ten-thousandth of a grain—the color produced will be rather more faint, though distinctly *orange.* (3) *Potassa* and *ammonia* yield with not very dilute solutions of brucia beautiful tufted crystals. (4) *Sulphocyanide of potassium* precipitates from a brucia solution an amorphous matter, which after a while assumes a beautiful crystalline form. Other reagents are *ferricyanide of potassium*, *bichloride of mercury*, *bichloride of platinum*, *carbazotic acid*, etc.

The application of brucia to frogs is followed by tetanic convulsions of precisely the same character as those occasioned by strychnia, the only difference being in the relative amount of the two alkaloids required to produce the result.

The *separation of brucia from organic mixtures* is effected in a manner similar to that employed for strychnia (*ante*, p. 428). If the nitric acid and chloride of tin test fails to produce the characteristic colors in the ultimate extract, the other tests will not succeed. Brucia has been detected in the *blood* of animals poisoned by it.

CHAPTER XXV.

ORDER III.—CEREBRO-SPINAL NEUROTICS.

DELIRIANTS.

THIS subdivision of the Neurotics has received the name of *Deliriants*, or *Delirifacients*, for the reason that active delirium constitutes one of their prominent symptoms in the human subject. They also occasion other symptoms in common, such as illusion of the senses and extreme dilatation of the pupil, heat and dryness of the throat, a flushed face, and frequently a redness of the skin and of the mucous lining of the throat. Occasionally there is irritation of the stomach and bowels, with dysuria, or suppression of urine. They all belong to the same natural order, *Solanaceæ*, and also to the same Linnæan class and order, *Pentandria Monogynia*. They comprise Belladonna, Stramonium, Hyoscyamus, and different species of Solanum. From their physiological property of dilating the pupil, they have also received the name of *Mydriatics*.

SECTION I.

POISONING BY BELLADONNA.—ATROPIA.

BELLADONNA (*Deadly Nightshade*).—The leaves, berries, seeds, and root of the *Atropa Belladonna*, or *Deadly Nightshade*, are all poisonous, and produce identical symptoms. The leaves and root are the parts used in medicine. Children are frequently poisoned from eating the berries.

The symptoms are as follows. A sense of heat and extreme dryness of the mouth and throat, with difficulty of swallowing, nausea, vomiting, giddiness, impaired vision, a flushed face, sparkling eyes, delirium of an excited maniacal character, spectral illusions, convulsions, followed by stupor and coma. The pupils are extremely dilated, and insensible to light. Cases have been reported where the pupils were contracted during sleep, but dilated in the waking state. Irritation of the urinary organs is not uncommon, such as strangury, suppression of urine, and hæmaturia. A scarlet eruption on the skin, resembling that of scarlatina, is frequently observed.

The *delirium* is of a peculiar character. The illusions are sometimes pleasing, exciting violent laughter; at other times they produce furious actions. There is loss of consciousness. The symptoms generally manifest themselves within one or two hours after swallowing the poison; but in poisoning from the berries they may be delayed for several hours. In cases of recovery, the symptoms are sometimes very long in disappearing.

The following case is quoted from the "New York Journal of Medicine," vol. viii. p. 284. A man ate a pie made with the berries of belladonna and apples. A few minutes afterwards he complained of feeling drowsy; the lethargy soon increased; his countenance changed color; the pupils became dilated, and he experienced a coppery taste in the mouth. On going up-stairs he staggered, and upon entering his room he fell, and became insensible. He subsequently became delirious and convulsed, and died the following morning. A child to whom a portion of the pie had been given died on the same day.

The following instance of recovery is related by Dr. Gray (N. Y. Jour. of Med., Sept., 1845, p. 182). A child between three and four years of age swallowed from eight to twelve grains of the extract of belladonna. About half an hour afterwards, the expression of the patient was that of terror; the pupils were widely dilated, and immovable; the conjunctivæ highly injected; and the whole eye prominent and very brilliant. The face, upper extremities, and trunk of the body exhibited a diffused scarlet efflorescence, studded with numerous papillæ, like the rash of scarlatina. The skin was hot and dry; pulse increased in force and frequency; respiration anxious and stridulous. There was a constant but unsuccessful attempt at deglutition, with spasmodic action of the muscles of the throat and pharynx; and paroxysms of violent motion, with rapid, automatic movements, attended with convulsive laughter. Under the action of an emetic, the alarming symptoms passed off in about three hours, and the child recovered, with the exception of a moderate diarrhœa, and a slight enlargement of the pupil.

The *external* application of belladonna, as also its administration as an enema, has occasioned serious and even fatal results. A case is related in which the injection of the decoction of the root caused death in five hours; and another, in which only two grains of the extract, administered in like manner, produced alarming symptoms. Dr. Lyman relates an instance in which the application of a small belladonna plaster to the chest of a nervous woman produced all the usual symptoms of poisoning by that substance, from which she did not entirely recover for four or five days. Cases in which a lotion of belladonna has produced similar results are mentioned in the "Chemical News" (Lond., Nov., 1866, p. 216).

The botanical characters of the leaves, fruit, and seeds of belladonna can usually be detected in the particles remaining in the alimentary canal after death. The *seeds* are very small, of an oval shape, and of a dark color. Under a low magnifier they present a honeycombed surface. The henbane-seed, which the belladonna-seed somewhat resembles, exhibits more irregular depressions. The purple coloring-matter of the berry is turned green by alkalies.

Atropia.—This alkaloid is the active principle of belladonna. It is a very powerful poison, producing symptoms essentially similar to those already described as resulting from belladonna, only more speedily. Symptoms of poisoning have been produced by the application of a weak solution of atropia to the eyes. One-eighth of a grain injected beneath the skin for the relief of sciatica, caused all the symptoms of belladonna-poisoning. Other instances are related in which much smaller quantities—even the one-hundredth of a grain—used hypodermically, produced alarming effects. Employed in this manner, in combination with morphia, its activity would appear to be in some manner modified. The author habitually uses, in his practice, a combination of one-fourth of a grain of morphia and about one-fortieth of a grain of atropia by hypodermic injection; and he has never witnessed any evidences of belladonna-poisoning resulting.

Fatal results may occur from the *external* application of atropia. Dr. Ploss, of Leipsic, reports a case in which an ointment composed of fifteen parts of sulphate of atropia to seven hundred parts of lard, applied as a dressing to a blistered surface on the neck of a man, caused death, under the most violent symptoms of belladonna-poisoning, within two hours. (Am. Jour. Med. Sci., April, 1865, p. 541.)

Fatal dose.—This has not been absolutely determined; but, from the known severe effects of doses less than one grain, it is highly probable that death would ensue from as small a quantity as *one-half to three-quarters of a grain*, provided the proper remedies were not applied. The following case is cited by Wormley from the "American Journal of the Medical Sciences," July, 1866, p. 269. A stout, healthy man swallowed from one-sixth to one-quarter of a grain of the alkaloid in solution. An hour afterwards, the patient was in a fearful state of excitement; the tongue was swollen, and projected between the teeth, and he incessantly moved it and his lips in a stammering manner, but without emitting a single intelligible sentence. The eyes were staring, the head hot, and the countenance livid; the pupils dilated to their utmost extent, and insensible to light. The pulse was rapid, full, and strong, and there was a constant but ineffectual attempt to urinate. During the following hour the excitement continually increased, when the subcutaneous injection of one-fifth of a grain of acetate of morphia into the right temple was soon succeeded by a state of calm. After two hours more, the excitement had again attained almost its former height; but it was again subdued by a repetition of the morphine injection. The patient now gradually recovered, the only symptoms remaining twenty-four hours after taking the poison being extreme weakness, dryness of the throat, slight twitching of the limbs, and dilatation of the pupils. Dr. Taylor also records a case from the "Medical Times and Gazette," July 6, 1865, p. 34,—that of a man who was very nearly killed by swallowing a grain of sulphate of atropia in solution. He did not fully recover for a fortnight; his pupils continued dilated for a week, and for several days there was partial paralysis of the bladder.

The criminal administration of atropia is a rare event. A trial for murder by this alkaloid took place at the Manchester Lent Assizes, 1872 (Reg. *v.* Steele). The prisoner, who was a nurse in a work-house, was charged with administering atropia to the senior surgeon, Mr. Harris, and thereby causing his death. The deceased was taken suddenly ill after his breakfast, and died with the usual symptoms of poisoning with atropia in about twelve hours. The poison was detected in the body by Mr. Calvert, and also in the liquid found in the room—a solution of atropia in spirit. Milk was the vehicle through which it was taken. As sent from the kitchen, this contained nothing injurious, but that found in the room of the deceased was tasted by two of the nurses, both of whom suffered from poisoning by atropia. Although the prisoner was proved to have had a strong motive for the murder, she was acquitted, from want of evidence as to the fact of putting the poison into the milk. (Taylor's Med. Jurisp., 1873, Am. ed., p. 251.)

The *diagnosis* of poisoning by belladonna or atropia is not always easy. The symptoms strongly resemble those caused by stramonium and hyoscyamus. The dilatation of the pupils—one of the most characteristic symptoms—may be occasioned by other causes. There seems to be a special tendency to the elimination from the system of the active principle (atropia) by the kidney. Prof. Guy states (Forens. Med., p. 512), on the authority of Dr. John Harley, that the presence of atropia in the urine can be readily proved within twenty minutes of the injection under the skin of one forty-eighth to one ninety-sixth of a grain, by the action of the urine on the eye. Twelve drops out of eight ounces of urine secreted in two and a half hours, while a patient is under the influence of one forty-eighth of a grain, will largely dilate the pupil, and will maintain it in that state for several hours.

Treatment.—The immediate evacuation of the stomach should be secured, either by a prompt emetic or by the stomach-pump. No *chemical* antidote is known, although various substances have been employed with this view, as tannin, animal charcoal, iodine in iodide of potassium, and hydrate of magnesia. The true *physiological* antidote is morphia, which has been repeatedly administered subcutaneously with the happiest result. Under the head of OPIUM, we have already remarked upon the antagonism between these two alkaloids in the human subject: each of them seems to be antidotal to the other, at least to a certain degree.

Morbid lesions.—These are not by any means characteristic of the poison. The vessels of the brain are more or less congested, and there are red patches in the pharynx and œsophagus, and at the cardiac end of the stomach. In some cases the whole gastro-enteric membrane has been found of a dark-purple color, probably dyed by the juice of the berries, and portions of the berries and some of the seeds have been discovered in the alimentary canal, or in the stools. The blood is usually liquid and dark-colored.

Chemical analysis.—When pure, atropia is in the form of white, crystalline tufts. Its taste is bitter and acrid. It is very slightly soluble in water, but very soluble in alcohol, ether, and chloroform. Heated on porcelain, it easily melts into a colorless liquid. It sublimes at 280° F. Its sublimate is less distinctly crystalline than either morphia or strychnia. Its color is not changed by either sulphuric, nitric, or muriatic acid. It has decided basic properties, neutralizing the strongest acids, and forming salts, several of which are crystallizable. Ammonia added to a solution of sulphate of atropia does not separate the alkaloid in distinct crystals, as in the case of morphia and strychnia.

Tannic acid precipitates the alkaloid from its solutions; but the most effectual precipitant, according to Winckler, is the *chloriodide of potassium and mercury*, which throws down a dense, white precipitate even in feeble solutions. By the use of this reagent he was able to determine the proportion of atropia contained in the powder of the dry leaves and root. In the leaves the alkaloid varies from 0.41 to 0.49 per cent., and in the root it amounts to 0.48 per cent. (Phar. Jour., June, 1872; quoted by Taylor.) Atropia is also precipitated by *chloride of gold;* but, unlike strychnia, it is not precipitated by sulphocyanide of potassium, or by chromate of potassa. An alcoholic solution of *carbazotic acid* produces in a solution of a salt of atropia a yellow, amorphous precipitate, which after a time becomes more or less crystalline, the crystals being in the form of transparent plates aggregated together. According to Wormley, one-thousandth of a grain will give a distinct crystalline deposit under the microscope when treated as above. This authority considers *bromine in hydrobromic acid* to be the most characteristic test for atropia. The precipitate at first is amorphous, and of a yellow color; but in a little time it becomes crystalline. It is quite insoluble in acetic acid, and very slightly so in either of the strong mineral acids. So small a quantity as one ten-thousandth to one twenty-five-thousandth of a grain will yield with this reagent a satisfactory result. (Micro-Chemistry of Poisons, p. 631.) Although this reagent produces yellow precipitates with the other alkaloids, yet they all, with the exception of that from meconin, remain amorphous; while the latter deposit differs from that of atropia in its form. This reagent likewise produces with *daturia* (the active principle of stramonium) a precisely similar precipitate; and this latter alkaloid is regarded by Wormley and others as being identical with atropia.

Examination of organic mixtures and the contents of the stomach.—The separation of atropia from complex organic mixtures is a different process. The one recommended by Prof. Wormley is a modification of the method of Stas, and is deserving of confidence. The solids having been properly cut up, the mixture is treated with an equal volume of strong alcohol, slightly acidulated with sulphuric acid, and gently heated for about half an hour. When cool, it is strained through muslin, the residue washed with alcohol, and all the liquids concentrated on a water-bath to a small bulk. If, during the evaporation, much insoluble matter separates, it is removed by a strainer. The cool, concentrated liquid is passed through a moistened filter, then transferred to a test-tube, and washed by agitating it with twice its volume of pure ether, which, after standing, is decanted and reserved for future examination. This washing with ether is again repeated—the object being to remove foreign matters. The aqueous solution is now rendered slightly alkaline by potassa, and thoroughly agitated with twice its volume of pure chloroform, which will dissolve the liberated alkaloid, if present. After complete separation of the liquids, the chloroform is removed, and allowed to evaporate spontaneously on a watch-glass. The evaporated residue should be dissolved in a few drops of water containing a trace of sulphuric acid, and then examined by the different reagents. The bromine test should first be tried on a single drop of the solution: if it fails to yield the characteristic reaction, the other tests may also be expected to yield negative results.

In case the bromine test gives a precipitate which *will not crystallize*, the remaining portion of the solution is diluted with a small quantity of water, then rendered alkaline by potassa, and the alkaloid again extracted with chloroform. On evaporating, the alkaloid is usually left in the crystalline state, which may be dissolved in a few drops of water and examined as before. Dr. Wormley was able, by the use of this method, to detect atropia in the stomachs of animals poisoned by comparatively small quantities of fluid extract of belladonna; and also in their blood (*loc. cit.*, p. 636).

The *physiological test* consists in the application of a portion of the ultimate extract to the eye, either of man or of one of the lower animals. Carnivorous animals are the most susceptible to its influence. According to Headland (Action of Medicines, p. 294), one three-thousandth of a grain, dropped in solution into the eye of an adult, will produce the characteristic effect. It must, however, be remembered that the other narcotics of this class will produce a similar impression.

SECTION II.

POISONING BY STRAMONIUM.—DATURIA.

The *Datura stramonium*, called also *Thorn-apple*, and *Jamestown* or *Jimson weed*, is a very common plant, abounding both in this country and in Europe. It grows very freely on the commons and waste grounds contiguous to towns. Other varieties are found in India and in other countries. All parts of the plant are poisonous, but especially the seeds and fruit. The seeds and leaves are employed in medicine. It owes its activity to a poisonous alkaloid named *daturia*.

Numerous instances of poisoning by stramonium have been recorded; but they have usually been the result of accident, and chiefly in cases of children, who have plucked and eaten the seeds.

Symptoms.—These are very similar to those produced by belladonna. There is the same dryness of the throat, with difficulty of swallowing; the dilated and insensible pupil; the incoherent and violent delirium; the headache, nausea, and vomiting; the blindness, ringing in the ears, and vertigo; the spectral illusions, and trembling of the limbs, followed by stupor and coma. Sometimes there are convulsions and paralysis, together with a scarlet eruption on the skin. Dr. H. Y. Evans, of Philadelphia, has reported (Amer. Jour. Med. Sci., July, 1866) an instance where seven children, aged from six to nine years, had each swallowed, it was stated, only ten of the seeds. Four hours after, the pupils of all were dilated to the utmost. In three of the children, who had swallowed the seeds without chewing them, the effects, which were slight, soon passed off; but in the four other cases, in which the seeds had been chewed, there were, in addition to dilatation of the pupils and perverted vision, confusion of intellect, deafness, intoxication, slow respiration, full pulse, and general loss of control over the muscles of the body. These symptoms were succeeded in a few hours by coma, and in one case by violent delirium. Emetics having failed to act, the stomach-pump was used. On the third day all remnants of the poisoning had completely disappeared, the pupils being the last to yield.

Dr. Calkins reports a case (Amer. Med. Monthly, Sept. 1856, p. 220) where a child, four years of age, swallowed over a tablespoonful of the seeds, and recovered after vomiting and purging, although they had remained in the stomach upwards of seven hours. Death may take place even although the whole of the seeds have been ejected, provided they have remained in the body for a sufficiently long period to allow of the absorption of the active principle. A case of this character is given by Mr. Duffin in the "Medical Gazette," vol. xv. p. 194,—that of his own child, aged two years, who swallowed about one hundred seeds of stramonium, without chewing them. In the course of an hour the usual symptoms were manifested, such as flushed face, dilated pupils, incoherent talking, wild spectral illusions, and furious delirium. In two hours and a half she lost her voice and the power of swallowing. The child died in twenty-four hours, although twenty seeds were ejected by vomiting, and eighty by purging.

In some instances the singular movements would appear to be owing to the perverted vision, which prevents the individual from properly appreciating the distance of objects.

The infusion of the *leaves* has likewise occasioned poisonous effects; as has also the alcoholic decoction of the seeds. An overdose of the extract—an officinal preparation—has produced death. Even the external application of the bruised leaves to the sound skin has given rise to the symptoms of poisoning; and Dr. Beck states (Med. Jurisp., ii. p. 877) that the bruising of the leaves in a mortar has caused dilatation of the pupil and irritation of the skin.

In India, the datura is much employed by the Thugs and other professional poisoners, not so much for the purpose of destroying life, as with the view of rendering the victims insensible, and consequently a more easy prey to robbery. According to Dr. Chevers (Med. Jurisp. for India, in Med. Times and Gazette, Feb., 1871), the Indian varieties are the *D. alba*, *D. fastuosa*, and *D. ferox*. Both the leaves and seeds are used, but particularly the latter, which are prepared by parching, decorticating, and pounding them, and in some instances by distilling from them an essence. The powder is mixed with curry or other food or drink of the intended victim, or given along with tobacco to smoke; while the essence is added to the sweetmeats so commonly used in the East. Death seldom results from the administration of the poison; but it is followed by very rapid insensibility, preceded by delirium. The effects vary according to the dose taken: sometimes stupor only is the result; at other times complete insensibility ensues. When the victim recovers from the immediate effects of the drug, he remains for some time in a fatuitous or delirious condition, often wandering about in a sort of intoxicated state, and completely oblivious of what had occurred.

Post-mortem appearances.—In some cases there is congestion of the brain, with effusion of bloody serum into the ventricles; patches of extravasation of blood in the stomach; marks of diffused inflammation over its lining membrane; congestion of the lungs; and the remains of the poisonous seeds in the stomach and intestines. In other cases nothing of an abnormal character has been discovered. It is quite certain that the *lesions* alone can lead to no conclusions as to the real cause of death.

The *treatment* is the same as that recommended for poisoning by belladonna. After the thorough evacuation of the poison, the hypodermic injection of morphia affords the most reliable means of relief: it should be persevered in until its effects are manifested by the contraction of the pupils.

Analysis.—The seeds are of a light-brown or black color, flattened, kidney-shaped, with a corrugated surface. They are much larger than those of belladonna or henbane. According to Prof. Guy, it requires one hundred and twenty henbane-seeds, and ninety of belladonna, to weigh one grain; but only about *eight* stramonium-seeds. There is no known *chemical* test to distinguish daturia from atropia. The only possible means of making the diagnosis is by identifying the seeds or portions of the leaves, by their physical and botanical properties.

Daturia.—This alkaloid is now regarded as being identical with atropia. The two are similar in appearance, chemical composition, solubilities, behavior with chemical reagents, and physiological action. The method of procuring daturia from the different preparations of the plant, as also from organic mixtures, including the contents of the stomach, the blood, and the urine, is precisely similar to that recommended under the head of ATROPIA, p. 441. The carefully-conducted experiments of Prof. Wormley on this subject would seem to leave no doubt as to the identity of the two alkaloids.

SECTION III.

POISONING BY HYOSCYAMUS.—HYOSCYAMIA.

The *Henbane* plant (*Hyoscyamus niger*) grows both in Europe and America. The *leaves* are officinal. The root is tapering, and somewhat resembles a small parsnip, for which it has been eaten in mistake. The *seeds* somewhat resemble those of belladonna, but are rather smaller: they are of a dark color, and thickly covered with ridges, easily recognized by a magnifier. Both the root and the seeds produce violent poisonous effects, which strongly resemble those of belladonna and stramonium. The preparations from the leaves (tincture and extract) are extremely variable in strength: frequently they are nearly, or quite, inert. The activity of the plant depends upon the soil, the period of its growth, the time of its collection, and the mode of preparation.

A curious instance of the effects of this narcotic is quoted by Taylor from the London "Lancet," July 6, 1844, p. 479. In a monastery, where the roots had been eaten for supper by mistake, the monks who partook of them were seized in the night with the most wonderful hallucinations, so that the place became like a lunatic-asylum. One monk rang the bell for matins at twelve o'clock at night; and of those of the fraternity who attended the summons, some could not read, some read what was not in the book, and some saw the letters running about the page like so many ants.

As regards the *fatal dose*, we have no positive data: the great uncertainty of the preparations of the drug as found in the shops, prevents any positive determination on this point. The dose of the officinal tincture is from half a drachm to two drachms; but Dr. Burder states that he has observed great inconvenience to follow from a dose of ten minims repeated every six hours (Lancet, July 6, 1844, p. 480). Dr. Cabot, of Boston, gave three teaspoonful doses of the tincture at intervals of an hour. Ten minutes after the last dose, the face began to swell, and became red and polished; the eyes were closed, and the patient could scarcely speak, on account of the swelling of the tongue and lips. The red discoloration of the skin extended as far as the umbilicus, and was attended with intolerable itching and burning (Am. Jour. Med. Sci., Oct., 1851). According to Wibmer, *twenty seeds* have produced complete delirium; and the same writer states that in one instance alarming symptoms were caused by seven grains of the *extract.* Fatal results have very rarely occurred. The use of a decoction of the plant as an injection has been attended with all the symptoms of apoplexy, with the exception of the stertorous breathing (Orfila).

Doubtless there may be an idiosyncrasy with respect to this drug, as in the case of opium. Dr. J. A. McFerran, of Philadelphia, related to the author the following instance which occurred in his practice. A man, aged about forty years, affected with a chronic cough, took two grains of the English extract of hyoscyamus. Within fifteen minutes he was completely narcotized; he had widely-dilated pupils, optical illusions, delirium, dry tongue and throat, inability to swallow, and disposition to stupor, so that he was obliged to be kept walking about to prevent his falling asleep. On the following day, his physician, scarcely crediting his story, administered to him one grain of the same extract; and within fifteen minutes the same symptoms were again manifested, though in a slighter degree.

Analysis.—When the vegetable has been eaten, it can be identified only by the physical and botanical characters of the portions discovered in the alimentary canal. *Hyoscyamia*, the active principle, is an alkaloid, which occurs in white, silky crystals, inodorous when pure, but, as usually prepared, having a disagreeable odor, like that of tobacco, and an acrid taste. It is very difficult to isolate. There is no characteristic *chemical* test for it. Its effect in causing dilatation of the pupil, when applied to the eye, would not distinguish it from either atropia or daturia. According to Dr. Harley, it speedily passes into the urine, after being swallowed; and it may be detected in this secretion by shaking it up with chloroform, evaporating the chloroform solution to dryness, and applying the extract to the eye.

SECTION IV.

POISONING BY SOLANUM.—SOLANIA.

Three species of the genus *Solanum* are usually referred to in the books as possessing poisonous properties: these are the *S. dulcamara*, Bittersweet, or Woody Nightshade; the *S. nigrum*, or Garden Nightshade; and the *S. tuberosum*, or the common Potato. They all contain a common active alkaloid principle—*Solania*, which in its physiological effects closely resembles the alkaloids existing in the other members of the class *Solanaceæ*.

The *Solanum dulcamara*, or Bittersweet, is a native of Great Britain, and is cultivated in our gardens. The dried stems are officinal. Its purple flowers and bright-red berries are sometimes eaten by children, on whom they act poisonously. They proved fatal to a boy four years of age, while two older sisters who ate them at the same time escaped with trivial symptoms. The fatal case was marked by vomiting and purging, convulsions and insensibility alternating, and death in convulsions in thirty-six hours after swallowing them. In two other instances, an unknown number of the berries proved fatal to two children. The dried stems, which are employed in medicine, possess extremely feeble narcotic properties.

The *Solanum nigrum*, or *Garden Nightshade*, produces white flowers and black berries. Its leaves and berries, when eaten by children, have given rise to symptoms of an acrid, narcotic nature. An instance cited by Tardieu, from M. Magne, exhibited the following effects. Two children about three and a half years old ate the leaves, and within two hours began to show signs of poisoning. One died in twelve hours, after having exhibited pain in the abdomen, with nausea, vomiting, and restlessness, followed by delirium. This increased to such an extent as to require restraint. The pulse was very quick, and scarcely perceptible, the respiration hurried, the face pale, and the pupils widely dilated. Convulsions of the limbs followed, which ended in coma and death. The child who survived was restless, frightened, and troubled with illusions, and the pupils strongly dilated. (Sur l'Empoisonnement, p. 755).

There is great discrepancy among authorities about the poisonous properties of the above two species of Solanum. This may possibly be ascribed to a real difference in the amount and in the strength of the poisonous principle, dependent upon the season and mode of growth. Some have supposed that the cases of poisoning that have been ascribed to these species were, in reality, to be accredited to the *Deadly Nightshade* (Belladonna), which had been mistaken for the others.

The berries and young shoots of the *Solanum tuberosum*, or common potato, have proved poisonous; and the berries have caused death, as in the case of a girl aged fourteen years, reported by Mr. Morris, in the "Lancet," June 28, 1858, p. 715. There were great restlessness and jactitation, and an anxious expression of countenance; the skin was cold, livid, and covered with a clammy perspiration; the pulse quick and very weak; respiration hurried; the jaws contracted; the speech lost; the patient constantly spat a viscid froth through the closed teeth. She died on the second day.

Sir R. Christison quotes from Dr. Kabler, of Prague, an instance where four persons in a family were seized with alarming symptoms, such as vomiting, coma, and convulsions, after eating potatoes that had commenced to sprout and shrivel.

Solania (*solanine*) is the active alkaloid principle common to all three of the above vegetables. When pure, it occurs in delicate, acicular, interlaced crystals. It is much less powerful in its action on man and animals than most of the other alkaloids. Schroff states that it has no influence over the iris.

Chemical properties.—It is nearly insoluble in water, very soluble in alcohol; sparingly soluble in absolute ether; very insoluble in chloroform. From aqueous solutions it is best extracted by hot amylic alcohol, or, better still, by a mixture of alcohol and ether. Either chloroform or ether may be employed to remove foreign matters from organic mixtures containing it.

Cold sulphuric acid immediately produces with solania an orange-yellow solution, which becomes brown on being heated. Nitric acid at first causes no change of color, but after a time it imparts a rose-red tint, which, on being heated, changes to a faint yellow (Wormley). The most characteristic *test* is cold, concentrated sulphuric acid. When a few drops of this are placed in contact with solania or its salts in the dry state, the deposit instantly assumes an orange-brown color, which slowly becomes an orange-yellow solution; in about an hour this acquires a purplish-brown color and throws down a brownish precipitate. After several hours, the solution becomes colorless, and the precipitate acquires a yellowish or dirty-white color (Wormley, *loc. cit.*, p. 663). Many reagents which precipitate the other alkaloids have no effect upon solania,—such as chloride of gold and platinum, iodide of potassium, ferrocyanide and ferricyanide of potassium, etc.

From organic mixtures solania is best recovered by adding a drop or two of sulphuric acid, and gently warming with half its bulk of alcohol. The cooled mass is then strained and evaporated on a water-bath, and again filtered; the filtrate is next evaporated almost to dryness, then stirred with a little pure water, and the solution filtered. This solution, which contains the *sulphate* of solania, may be decomposed by *hydrate of lime*, and the solania separated from the sulphate of lime by warm alcohol, and this evaporated to dryness. (Wackenroder; quoted by Wormley.)

CHAPTER XXVI.

DEPRESSANTS.

UNDER this subdivision are conveniently arranged several very active Neurotic poisons, which agree in the property of causing great depression of the muscular system, although in some other respects they may differ from one another.

In thus grouping together different poisons it is not intended to imply that they all possess one identical physiological mode of action. The substances that will be considered under this subdivision are Hemlock (*Conia*); Tobacco (*Nicotina*); Lobelia; Aconite (*Aconitia*); and Calabar bean (*Physostigmia*).

SECTION I.

POISONING BY HEMLOCK (CONIUM MACULATUM).—CONIA.

The spotted hemlock of Great Britain and America is believed to be the same plant as the *Cicuta* of the ancient Greeks, the one that furnished their celebrated state poison. It belongs to the natural order *Umbelliferæ*, which also includes several other very poisonous plants,—the *Æthusa cynapium*, or Fool's parsley, the *Cicuta virosa*, or water hemlock, and the *Œnanthe crocata*, or hemlock water-dropwort.

Every part of the Conium maculatum possesses poisonous properties, and emits a peculiar, disagreeable, mousy odor, which becomes more perceptible when it is bruised in a mortar along with a solution of potassa. This odor depends upon its volatile active principle. The leaves and fruit are used in medicine in the form of *succus* (which is procured from the fresh leaves), and extract. The former is the only reliable preparation.

Poisoning from hemlock is nearly always the result of accident, the leaves having generally been eaten in soup by mistake for parsley, which it somewhat resembles. The accounts of its action upon the human system are somewhat contradictory. Some authors ascribe to it positive narcotic properties, while others, who have experimented with it upon themselves, deny that it possesses any soporific power, whilst they experienced dryness of the throat, headache, a numbing, pricking sensation gradually extending up the limbs, and almost amounting to temporary paralysis. The eyes were likewise affected, giving the sensation of objects obstructing the vision. Dr. John Harley experimented upon himself with five and a half drachms of the *succus*. The effects were general muscular lethargy, loss of muscular power, particularly of the eyelids, which were kept open with difficulty; the pupils were dilated. The mind remained unaffected. (Phar. Jour., 1867.)

The *post-mortem appearances* in the few fatal cases noticed were those of apnœa, with redness of the mucous membrane of the stomach, and congestion of the brain—signs of no characteristic significance. Sometimes the remnants of the seeds or leaves may be identified by the microscope, and by rubbing them in a mortar with liquor potassæ, when the peculiar mousy odor will be developed.

According to Dr. Harley, its influence appears to be in proportion, not to the muscular strength, but to the motor *activity* of the individual; so that a person of active, lively habits is much less affected by it than one of indolent, sluggish disposition. Thus, "an active, restless child will often take with scarcely any appreciable effect a dose sufficient to paralyze an adult of indolent habits." It seems to have no effect upon the cerebrum, since even when given in fatal doses, "while the eyes will be completely fixed and the pupils dilated, while all power of motion is lost, and the individual appears to be in the most profound coma, the perceptive faculties and reasoning powers may be as acute as ever." (The Old Vegetable Neurotics, 1868.) Death appears to be the result of paralysis of the muscles of respiration, including the diaphragm. It is generally preceded by convulsions. The heart has continued to beat after all other signs of life had ceased.

Conia.—This alkaloid, known also under the name of *conine* and *conicine*, exists most abundantly in the *seeds* of the plant. It is one of the most actively fatal poisons known, almost equaling prussic acid in this respect. Sir R. Christison states that a single drop of the alkaloid applied to the eye of a rabbit killed it in nine minutes; and three drops applied in the same manner killed a strong cat in a minute and a half. Five drops put into the throat of a small dog began to act in thirty seconds, and proved fatal in one minute; and two grains of the chloride injected into the femoral vein of a young dog killed it before there was time to note the interval. (On Poisons, p. 655.) In Prof. Wormley's experiments, a single drop being placed upon the tongue of a large cat, the animal was inclined to stand still, and manifested an unsteady gait when disturbed; in two minutes and a half it fell upon its side, voided urine, had violent convulsions of the limbs, with trembling of the body; and it died in three minutes from the time of the administration of the poison. In another animal, the pupils became dilated and immovable, the legs became powerless, and death occurred in four minutes, being preceded by violent convulsions.

Treatment.—Prompt emesis by mustard and water; active stimulation, both external and internal. Pereira suggested strychnia as a physiological antidote, on account of its opposite effects on the system.

Chemical properties.—Conia, in its pure state, is a colorless, transparent, volatile, oily liquid, having a strong alkaline reaction; its odor is peculiar, repulsive, and suffocating, resembling that of a stale tobacco-pipe. When diluted with water it emits an odor resembling that of mice. This peculiar odor is perceptible even when it is diluted with fifty thousand times its weight of water (Wormley). It gives a greasy stain to paper; burns with a bright, smoky flame; its taste is disagreeable and persistent; it boils at about 350° F., but it distills over with the vapor of water. It speedily changes when exposed to the air, becoming yellowish, then brownish, and is finally resolved into a resin and ammonia. It is partially soluble in water, very soluble in alcohol, ether, and chloroform: the two latter will separate it from its aqueous solutions.

Tests.—If a drop of the alkaloid be placed in a watch-glass, and covered by another glass holding a drop of hydrochloric acid, both glasses immediately become filled with dense white fumes, and the drop of conia soon becomes a mass of beautiful, delicate, crystalline needles, which are permanent in the air. Even diluted solutions of the alkaloid will yield similar white fumes when exposed to the acid vapor, which are followed by the formation of crystals on concentration. Sulphuric acid gives with it a pale-red solution, which after a few days deposits crystalline needles. Nitric acid causes with it dense white fumes; it deepens its color, and ultimately deposits crystals. Strong hydrochloric acid imparts to it a pale-red tint, which gradually becomes much deeper: on evaporation, needle-shaped crystals are deposited. Oxalic acid forms with it prismatic crystals of the oxalate of conia. Like the fixed alkaloids, it yields precipitates with tannic acid, iodo-iodide of potassium, terchloride of gold, corrosive sublimate, carbazotic acid, and some other reagents; but not with bichloride of platinum, iodide of potassium, ferrocyanide and ferricyanide of potassium, or bichromate of potassa. Its liquid, oily condition, together with its peculiar odor, will serve to distinguish it from all other substances except *nicotina:* the points of difference between these two alkaloids will be mentioned under the head of *Nicotina.*

Detection in organic mixtures.—Conia may be separated from organic mixtures, as the contents of the stomach, the urine, or the blood, by the process of M. Stas, somewhat modified. Wormley recommends to exhaust the material with dilute acetic acid, as in the process for nicotina (see *post*, p. 462). Dr. Harley macerates the suspected substance for a few days in water acidulated with one-fiftieth of its bulk of sulphuric acid; this should be evaporated to a syrup, the residue mixed with an equal bulk of a strong solution of potassa, transferred to a long tube, and agitated with its bulk of ether several times during twenty-four hours. The ether is then decanted, and the residue washed several times with fresh ether. On distillation of the ethereal solution, impure conia remains. This is to be shaken with a small quantity of dilute sulphuric acid, which separates the alkaloid from the impurities. From this solution of sulphate of conia the base is separated in the usual way, viz., by evaporation, mixing with caustic potassa, and shaking with ether; and then evaporating, to obtain the characteristic oily globules, which should respond to the several tests above mentioned.

Dr. Harley gives a salutary caution in regard to relying too strongly on what is supposed to be the characteristic *odor* in the search for conia in organic mixtures, especially the urine. He remarks: "In examining the animal fluids or tissues for conia, we must bear in mind that the addition of caustic potash to them will often develop an odor indistinguishable from conia; and that nothing short of the isolation of the principle itself should satisfy us" (*loc. cit.*, p. 19). He also relates a case in which he could not distinguish an ethereal extract, obtained from a patient's urine who had not taken conia, when treated with potassa, from an aqueous solution of conia, used for comparison. Dr. Taylor, speaking of the same fallacy, says that "an incautious operator may readily come to the conclusion that he has found 'traces,' and ascribe death to the poison." He then cites an instructive case that occurred in Germany, where a man died in a few hours after going to bed, and it was alleged that his wife had poisoned him. Those who examined the body deposed that they had found traces of conia in the stomach, intestines, and kidneys; and the wife was accused of having administered hemlock. Some doubts having arisen in the minds of the authorities, the matter was referred to Mitscherlich and Casper, who found that the chemical process pursued failed to detect conia in the body; that there was nothing to indicate that the deceased had taken hemlock in any form; but that, on the contrary, after having eaten and drunk freely, he had vomited after going to bed, and a portion of the food had entered the trachea and had suffocated him. (Med. Jurisp., Amer. ed., 1873, p. 242.)

The other hemlocks—*Cicuta virosa,* or water hemlock, *Œnanthe crocata*, or *hemlock water-dropwort*, and *Æthusa cynapium*, or *Fool's parsley*, or *lesser hemlock*—are all extremely poisonous, producing symptoms of an acrid, narcotic character. The Œnanthe is particularly dangerous, being, in fact, one of the most poisonous of the umbelliferous plants. No alkaloid has as yet been isolated from any of those just mentioned.

SECTION II.

POISONING BY TOBACCO.—NICOTINA.

The dried leaves of the *Nicotiana tabacum*, a plant belonging to the natural order *Solanaceæ*, constitute the well-known tobacco so universally employed throughout the world. It owes its activity and poisonous properties to a volatile liquid alkaloid of an oily consistence, *nicotina*, which somewhat resembles conia, and which exists in different proportions in different specimens of the leaves, varying from two to seven or eight per cent.

Symptoms.—A large dose of tobacco (or even a small one in those unaccustomed to its use) produces very decided symptoms. Very soon after taking it, the individual experiences vertigo, sense of confusion of the head, nausea, vomiting, severe retching, heat in the stomach, great anxiety, excessive prostration, cold, moist skin, trembling of the limbs, and sometimes severe purging. The pulse is small, weak, and scarcely perceptible; there is difficulty of breathing, and involuntary urination. In some cases there is violent pain in the abdomen; in others, there is rather a sense of sinking or depression in the region of the heart, passing into syncope, and a feeling of impending dissolution. The pupils do not seem to be always similarly affected. Dr. Taylor states that they are dilated; whilst Dr. Pereira (Mat. Med., ii. p. 494) speaks of tobacco as differing from belladonna and stramonium, in causing contraction of the pupils, both when applied directly to the eye, and when taken internally; and also by the absence of delirium, and of dryness of the throat. Wharton and Stillé (Med. Jurisp., 1873, vol. ii. p. 609) state that the pupils are but slightly affected, and that they preserve their sensibility to the light. Death is often preceded by convulsions and paralysis.

The *external* application of tobacco to abraded surfaces, and even to the healthy skin, will occasion severe and sometimes fatal consequences. Tardieu mentions several instances where decided symptoms were produced by the application of the dried leaves to the naked body (Sur l'Empoisonnement, p. 780). A decoction of tobacco applied by a man for the cure of an eruptive disease, caused death in three hours. (Am. Jour. of Med. Sci., Jan., 1865, p. 268.)

Its fatal effects when administered by the rectum are well known to physicians. Pereira mentions a case where half a drachm, given in the form of an enema, caused death; and Dr. Tavignot witnessed a case in which *fifteen grains* thus administered produced a fatal result in a robust man, aged fifty-five years (Rev. Méd., Nov., 1840). Even tobacco-smoke, diffused through water and swallowed, has caused the death of a young infant. (Wharton and Stillé, *loc. cit.*, p. 610.)

The *smoking* of tobacco has been known to produce violent and even fatal effects. Two instances of the latter are related by Gmelin, as resulting from excessive smoking, in one case of seventeen, and in the other of eighteen, pipes of tobacco at a sitting.

The rapidity of the action of tobacco on the human system varies with the dose and the mode of application. In one case, an unknown quantity of snuff swallowed in whisky caused death in an hour. In another case, quoted by Beck (Med. Jurisp., ii. p. 878), an enema of tobacco used by a female for the expulsion of worms, produced violent convulsions, and death in *fifteen minutes.* In another case, quoted by Christison, a tobacco enema caused death in thirty-five minutes. The application of *nicotina* to the tongue of an animal has caused death *within two minutes.*

Post-mortem appearances. — No special anatomical lesion characterizes this poison. A diffused redness of the omentum and of the stomach and bowels, with patches of extravasation in the mucous membrane, together with an empty state of the heart and blood-vessels of the abdomen, is about all that has been observed. If the leaf or powder has been swallowed, these may be recognized by their physical and botanical characters when examined microscopically. In a case reported by Dr. Taylor, in which death resulted in seven hours from swallowing an ounce of crude tobacco, the brain and upper portion of the spinal marrow were congested; the heart was empty, small, and contracted; the liver and kidneys much congested; the intestines contracted throughout; the mucous membrane reddened and partially abraded.

The bladder was empty and contracted; the blood was liquid and dark-colored. No peculiar odor was perceived.

In a case of suicidal death from *nicotina*, occurring in London in 1858, and examined by Dr. Taylor, the post-mortem appearances were general relaxation of the muscular system, staring eyes, bloated and livid features, the vessels of the scalp and membranes of the brain, and those of the lungs, gorged with black blood, and the cavities of the heart, with the exception of the left auricle, empty. There was intense congestion of the mucous membrane of the stomach, and of the liver. The blood was black and liquid, and in some parts had the consistence of treacle. No peculiar odor was perceptible. (On Poisons, p. 661.)

NICOTINA (*Nicotine*).—This alkaloid, when pure, is a colorless, oily liquid, which assumes a light yellowish tint on exposure to the air, and deepens and thickens by keeping. It produces a greasy stain on paper, like conia, which disappears on exposure. It is usually described as possessing an acrid, unpleasant odor: this is true of the samples generally found in the shops, but in a perfectly pure specimen the odor is ethereal and pleasant. Prof. Guy states that this odor has been retained for several years in two specimens in his possession. It has a strong alkaline reaction, and a density of 1.048.

It is freely soluble in water, and even a very dilute aqueous solution will retain the peculiar odor. It is also soluble in alcohol, ether, chloroform, the fixed oils, and in oil of turpentine. Either chloroform or ether may be employed to extract it from its solutions in water. Its taste is very pungent and acrid, even when much diluted, producing a peculiar sensation in the throat and air-passages. It slowly distills at about 295° F., and boils at about 470°. It may be distilled unchanged in an atmosphere of hydrogen gas. Heated on platinum or on paper, it burns with a bright flame, emitting a thick, black smoke.

Nicotina is one of the most rapidly-fatal poisons known: it rivals hydrocyanic acid in this respect. A single drop destroyed a rabbit in three and a half minutes. In fifteen seconds the animal lost all power of standing, was violently convulsed in the legs and back, the latter being arched (*opisthotonos*). (Taylor's Med. Jurisp., Am. ed., 1873, p. 227.) In Wormley's experiments, one drop put into the mouth of a full-grown cat produced *immediate* prostration, continued convulsive movements of the extremities, and death in *seventy-eight* seconds. Another cat died from a similar dose, and with similar symptoms, in *seventy-five seconds* after taking the poison.

In the celebrated case of the *Count Bocarmé*, who was executed in Belgium, in 1851, for poisoning his brother-in-law, Gustave Fougnies, nicotina was the agent used. An unknown quantity was forcibly put into the throat of the victim, the countess assisting her husband as an accomplice in the murder. Death was believed to have taken place within five minutes. The poison was detected by M. Stas in the tongue, throat, stomach, liver, and spleen of the deceased, and also from stains on the floor near where the act was committed. From the admirable and exhaustive report of the examination of the body by M. Stas, we may note the following particulars. The appearance of the tongue indicated the action of some highly acrid agent: it was swollen, blackened, softened, and friable; the epithelium was detached with facility. This was also the condition of the mucous lining of the mouth and pharynx: it was reddened as if cauterized, and was removed with the greatest ease by the handle of the scalpel. The lining membrane of the stomach was intensely injected, exhibiting large patches, which were livid and black. The vessels were filled with a black coagulum, which resembled blood that had been treated with strong sulphuric or hydrochloric acid. The duodenum was also highly injected. There were no ulcerations or perforations of the stomach or bowels. The lungs were gorged with black blood, and exhibited the usual characters of asphyxia. The heart was normal; its cavities contained black blood, not coagulated. No mention is made of any peculiar odor being noticed in the body. (For a fuller account of this interesting case, see Orfila's Toxicologie, ii. p. 498; also, Wharton and Stillé's Med. Jurisp., 1873, ii. p. 612.)

Chemical reactions.—If a drop of nicotina be put into a watch-glass, and this covered with another glass, inverted, containing a drop of either hydrochloric or nitric acid, the glass will become filled with white fumes: these are not so dense as those produced by conia under similar circumstances, nor do they give rise to the formation of crystals. The strong acids, when applied directly to it, produce no characteristic effect.

Nicotina unites freely with acids, forming salts, which retain the peculiar taste of the alkaloid, but are destitute of odor. They are mostly soluble in water and alcohol, but not in ether or chloroform. If a salt of nicotina be distilled with a caustic alkali, the free alkaloid will be found in the distillate, and usually associated with ammonia. If the distillate thus obtained be neutralized by oxalic acid, then gently evaporated to dryness, and the residue treated with alcohol, the oxalate of nicotina will be dissolved, while the oxalate of ammonia will remain undissolved, and may be separated by filtration. The alcoholic solution, on evaporation, will yield pure oxalate of nicotina.

(1) *Bichloride of platinum* produces in an aqueous solution of nicotina a yellow, turbid precipitate, which ultimately assumes a crystalline form, well marked under the microscope, and of a character totally distinct from that of the double chloride of platinum and potassium, or ammonium.

(2) *Corrosive sublimate* throws down a copious white, curdy precipitate, which soon acquires a yellow color, and deposits beautiful groups of colorless crystals, which are permanent in the air. In very dilute solutions of nicotina, this reagent at first causes a turbidness, but after a time, especially on stirring with a glass rod, the peculiar crystals appear, which resemble flowers, winged insects, and rosettes. Although corrosive sublimate produces white precipitates with ammonia and with many of the alkaloids, yet all these deposits, unlike that of nicotina, remain amorphous, except the precipitate from strychnia; and in this case the crystals are wholly unlike those obtained from nicotina. Prof. Wormley considers this to be the most valuable test yet discovered for nicotina (Micro-Chem. of Poisons, p. 433).

(3) *Carbazotic acid* yields with aqueous solutions of nicotina a yellow, amorphous precipitate, which ultimately becomes a mass of yellow, crystalline tufts, to be identified by the microscope.

(4) *Iodo-iodide of potassium* yields a brownish-red, or yellow, amorphous deposit, which, after a time, may entirely disappear, to be at once brought back on further addition of the reagent. A similar result is produced with most of the alkaloids: hence it is not a *characteristic* test. But as its reaction is extremely delicate for nicotina, serving to detect even less than the one-hundred-thousandth of a grain, it is obvious that, if a suspected solution of nicotina fails to yield a precipitate with this test, it will be useless to expect any results from the other reagents.

(5) *Terchloride of gold* produces a yellow, amorphous precipitate, even in very dilute aqueous solutions of nicotina, in common with many of the other alkaloids. The same is true of *bromine in hydrobromic acid.*

As ammonia gives very similar reactions with some of the above reagents, it is important to be able to distinguish between them. *Tannic acid* gives a white, amorphous precipitate with nicotina, but merely imparts a red color to ammonia. Gallic acid yields no precipitate with nicotina; with ammonia it produces a pinkish-red color, rapidly changing to an olive-green. Iodine-water gives a brown precipitate, while with ammonia there is no precipitate, but the color is discharged.

Separation from organic mixtures, or the contents of the stomach.—The process already described in the former part of this work (p. 110) as the process of Stas, is especially adapted for the recovery of nicotina from organic mixtures. In fact, it was the very process employed by its originator in the Bocarmé case, above alluded to. Other good authorities have somewhat modified the original process. Wormley exhausts the suspected material with water instead of alcohol, acidulated with *acetic acid;* Taylor employs *sulphuric acid;* Stas originally used *tartaric acid.* After proper maceration, filtration, and concentration, the residue containing the nicotina salt may either be neutralized by caustic potassa, and distilled over; or (as is most usual) it is rendered alkaline by potash or soda, and thoroughly shaken in a stout tube, with about two volumes of chloroform, or about five volumes of ether, and the mixture allowed to repose until the fluids have completely separated. The chloroform (or ether), having been carefully separated, is allowed to evaporate spontaneously on a watch-glass, when any nicotina present will be left in the form of oily streaks or drops, having the peculiar odor of the impure alkaloid, which is made more evident by heating gently. This should be corroborated by making a solution with a very small quantity of pure water, and applying the above-mentioned reagents, using first the corrosive sublimate test. A drop of the solution may also be put within the beak of a small bird, or in the mouth of a rabbit.

Nicotina may also be readily detected in the tissues and in the blood, in the absorbed state: the process to be employed is essentially the same as that above described. M. Stas was probably the first to show that it is possible to recover an absorbed alkaloid from the tissues. About the same time, Orfila procured the same poison (nicotina) from the liver and spleen of dogs killed by this substance (Toxicologie, ii. p. 493).

For a full detail of the mode of separating nicotina from organic mixtures, the reader is referred to Wormley's "Micro-Chemistry of Poisons."

POISONING BY LOBELIA.—LOBELINA.—The *Lobelia inflata*, or *Indian tobacco*, is a native of North America. It is extensively used both in this country and in Great Britain as the standard remedy by a set of quacks denominated *Thomsonian*, or *Botanical doctors*. According to Dr. Letheby, thirteen cases of poisoning by this substance had occurred in England within three or four years; and Dr. Beck states that "thousands of individuals in the United States have been murdered by the combined use of capsicum and lobelia administered by the Thomsonian quacks" (Med. Jurisp., vol. ii. p. 736). The leaves and seeds are the parts of the plant employed. They owe their activity to a fixed alkaloid named *lobelina*.

Effects on the system.—In small doses, lobelia acts as an expectorant; in larger doses, as a powerful emetic and depressant. In poisonous doses, it produces distressing nausea and vomiting, sometimes purging, copious perspiration, extreme relaxation, anxiety, prostration, very feeble pulse, contracted pupils, insensibility, occasionally convulsions, and death. A drachm of the powdered leaves has occasioned death.

The *post-mortem appearances* that have been noticed were inflammation and softening of the mucous membrane of the stomach, and inflammation of the bowels. The vessels of the brain are sometimes strongly congested. When employed by enema, it occasions very much the same alarming and fatal depression of the system as tobacco.

Lobelina (*lobeline*) is a yellowish liquid, lighter than water, of somewhat aromatic odor, and very acrid, persistent taste. It is soluble in water, but more so in alcohol and ether: the latter readily separates it from its aqueous solutions. It has an alkaline reaction, forming soluble salts with the acids. A boiling heat decomposes it. Tannic acid immediately precipitates it from its solutions. It resembles nicotina in many of its properties, just as lobelia resembles tobacco. By experiments on animals, lobelina seems to produce the narcotic, but not the emetic, effects of the plant. (See paper by Prof. Procter, in American Journal of Pharmacy, ix. p. 105, and xiii. p. 1.)

In a case of death from the use of lobelia, the diagnosis would be materially aided by the discovery of fragments of the leaves, or the seeds, in the alimentary canal, which might be identified by the microscope. No case has been recorded, so far as we are aware, of the administration of lobelina with either a homicidal or a suicidal intent. (For the report of two interesting trials for fatal poisoning by lobelia, under the "botanical" treatment, see Wharton and Stillé's Med. Jurisp., 1873, vol. ii. pp. 586 and 963.)

SECTION III.

POISONING BY ACONITE.—ACONITIA.

The *Aconitum napellus* (Monkshood, or Wolfsbane) is indigenous in Europe, and is cultivated in our gardens. It belongs to the natural order *Ranunculaceæ.* All parts of the plant are poisonous, but the root is the most so. The root and leaves are officinal, and are easily identified by their appearance. The root has occasionally been fatally mistaken for *horseradish* root; but their characters are totally distinct.

The root of the aconite is conical, rapidly tapering to a point, and throwing out numerous curling fibres; it has a dark-brownish color; and when a fragment of it is chewed it imparts to the lips, tongue, and fauces a peculiar tingling, numbing sensation, which is quite persistent. The horseradish root, or stick, is cylindrical, and truncated, not conical; its color is whitish; and when chewed, as is well known, it leaves merely a sweetish, pungent impression, totally distinct from that of aconite root.

Aconite root has been administered with criminal intention in at least one recorded case, where the powdered root was mixed with pepper, and sprinkled over the greens used for dinner by the deceased (Dublin Jour., July, 1841).

Symptoms.—On animals.—According to Dr. Fleming, aconite, when introduced into the system of one of the lower animals, produces, successively, weakness of the limbs and staggering; accelerated or laborious breathing; paralysis; diminution, or total loss, of sensibility of the surface; increasing difficulty of breathing; and, after a few spasmodic twitches, death by asphyxia. In a few instances there were decided convulsions, and even opisthotonos; the pupils were generally *contracted* (this is contrary to the experience of Headland in the case of animals, and also differs from the recorded cases of poisoning in man, in all of which the pupils were more or less *dilated*). On opening the body after death, the heart was found beating with considerable strength; there was great congestion of the venous system, with distension of the right side of the heart.

On man.—There is a sense of burning, tingling, and numbness of the mouth, throat, and stomach, followed by nausea and vomiting, with pain and tenderness of the epigastrium. The numbness and tingling speedily become general, with diminution of the sensibility of the surface, vertigo, dullness of vision, or complete blindness; tinnitus aurium, with occasional deafness; frothing at the mouth; sense of constriction of the throat and of weight at the stomach; great muscular weakness; a slow, feeble pulse; difficulty of breathing; cold, clammy skin; blanched countenance; perhaps a few convulsions, and death. The mind throughout seems to be unaffected, the patient retaining consciousness to the last. The brain appears entirely unaffected; there is no tendency to sleep or to coma. In a few exceptional cases, death is preceded by delirium and convulsions. Death is apt to supervene suddenly. In fifty-three cases of aconite-poisoning collected by Dr. Tucker, of New York (N. Y. Jour. of Med., March, 1854), cited by Wharton and Stillé, general convulsions occurred only in seven, and delirium and stupor only in three. In seventeen out of twenty cases the pupils were dilated.

Post-mortem appearances.—The autopsy reveals nothing characteristic. There is usually considerable engorgement of the vessels of the brain, and likewise of the lungs and liver. There is sometimes redness of the mucous membrane of the stomach and bowels, which are frequently found empty. The blood is generally fluid, and of a dark color. These are merely the usual attendants on death from asphyxia; and from the fact that in animals poisoned by aconite the heart has been found beating after death, it would seem as if this poison destroys life by *asphyxia.* In some cases, death appears to be due to *syncope.*

The *quantity* necessary to destroy life is undetermined. This probably arises from the variable strength of the preparations of aconite, as found in the shops. These preparations are the *tinctures* of the leaves and roots, and the alcoholic *extract.* The latter especially is apt to be very inert. Sir R. Christison gave six grains of a carefully-prepared extract to a woman suffering with rheumatism, without visible effect

(On Poisons, p. 667); whilst Dr. Fleming speaks of two grains producing alarming effects, and of four grains proving fatal. One drachm of the tincture has caused death on several occasions. The case related by Dr. Easton (Glasgow Med. Jour., July, 1853), in which twenty-five minims of the tincture were taken, shows probably the smallest dose that has proved fatal. Wharton and Stillé mention an instance where twenty-five drops proved fatal in four hours, in the case of a gentleman who took it by mistake (Med. Jurisp., ii. p. 629). An excise officer in England died in a few hours after merely tasting Fleming's strong tincture: he had swallowed probably not over a teaspoonful. Dr. Pereira speaks of a case where two doses of six drops each, taken at an interval of two hours, produced most alarming symptoms in a young man (Mat. Med., ii. p. 1091); and Dr. Wormley alludes to a case falling under his own observation, in which five drops of Thayer's fluid extract of the root produced most decided symptoms of poisoning, with alarming prostration, which continued for about two hours (*loc. cit.*, p. 611).

On the other hand, as in the case of the other violent poisons, recoveries occur after swallowing very large doses. In such cases, most of the poison has been removed through prompt and active emesis. In fatal cases, death generally occurs within two or three hours; though sometimes life is prolonged for twenty-four hours.

ACONITIA (*Aconitine*).—This alkaloid is the active poisonous principle of aconite. It abounds most in the root, although its proportion here is only from one-tenth to one-fifth of one per cent. (Wormley). In its *pure* state, it is probably the most violent poison known: Pereira states that *one-fiftieth of a grain* nearly proved fatal to an elderly lady (*loc. cit.*, 1093). Much of the aconitia sold in the shops is almost, if not entirely, inert. Dr. Pereira swallowed one grain of a French preparation without experiencing the slightest effect; and Wormley says that of three German specimens examined by him, two were entirely inert, and the third nearly so. The only reliable article, heretofore, has been that prepared by Morson, of London.

Treatment.—There is no known chemical antidote. The poison should immediately be evacuated from the stomach by the use of active emetics or the stomach-pump. Stimulants should be freely used both outwardly and inwardly. Finely-powdered animal charcoal, mixed with water, has been recommended by Headland and others, as also tannin, or astringent infusions. Dr. Wormley cites the case of a child, five years old, to whom the tincture of nux vomica was administered as an antidote to the tincture of aconite, on physiological principles, after unavailing efforts to excite vomiting had been employed. The effect of the first dose of the antidote was to increase the force of the heart's action and the strength of the respiration. A second dose of tincture of nux vomica (three drops) was administered at the end of twenty minutes, which was followed by vigorous vomiting, and a speedy recovery (Amer. Jour. Med. Sci., Jan., 1862, p. 285). Dr. Fleming recommends friction to the spine and limbs by means of warm cloths; and for the great dyspnœa and extreme feebleness of the heart's action, slight galvanic shocks to be passed through the heart, and the employment of artificial respiration.

It would appear that *digitalis* has the power of acting as a physiological antidote to aconite. It was discovered by Dr. J. Milner Fothergill (Digitalis, Lond., 1871) that where digitalis is administered to frogs under the influence of aconite, the heart is visibly relieved from the depression produced by the first poison. Even when all the cardiac action had apparently ceased, digitalis had power to recall the systolic movements, until, finally, a return to the normal state was brought about. A case is reported in the "British Medical Journal" of December 11, 1872, where recovery took place after swallowing an ounce of Fleming's tincture of the root. The patient when first seen was apparently dying. Twenty minims of the tincture of digitalis were injected hypodermically, and after twenty minutes the man had revived sufficiently to swallow; a fluidrachm of the tincture was given along with brandy and ammonia, and was twice repeated within an hour. (Dr. H. C. Wood's Therapeutics.) The above facts certainly warrant the trial of digitalis in a case of aconite-poisoning.

Pure aconitia occurs as a white, transparent, granular solid (it has lately been procured in crystals). Its taste is at first acrid, soon followed by a feeling of tingling and numbness of the lips and tongue. Its solution applied to the skin occasions a feeling of heat and numbness. So active is this poison that, according to Dr. Headland, one three-hundredth of a grain will kill a mouse; one-twentieth of a grain, a cat; and one-tenth of a grain, a man. One-thousandth of a grain causes tingling and numbness on the tip of the tongue; and one-hundredth of a grain dissolved in spirit and rubbed into the skin, causes loss of feeling, lasting for some time (Guy's Foren. Med., p. 566).

Aconitia is unchanged by exposure to the air. Heated on porcelain, it fuses into a yellow liquid, which gradually becomes darker, and finally is reduced to carbon. It has strong basic properties, forming salts with the acids, several of which are crystalline. It is very slightly soluble in water; quite soluble in alcohol and chloroform; less so in ether. Its *salts* are very soluble in water and alcohol, but insoluble in ether. None of the strong mineral acids causes any change of color in it, when cold; but sulphuric acid when warmed imparts to it a *brown* tint; heat causes no change with the other acids. There is not a single chemical test that is *characteristic* of aconitia. The alkalies, carbazotic acid, chloride of gold, iodo-iodide of potassium, and bromine in hydrobromic acid, all yield precipitates with a solution of aconitia, but nothing of a peculiar character. Bichloride of platinum, the chromate of potassa, and ferrocyanide and ferricyanide of potash, do not precipitate it. Its presence can be established (in a medico-legal case) only by the physiological test,—*i.e.* the peculiar tingling sensation imparted to the tongue and lips on the application of a minute portion of the ultimate extract obtained from the suspected material, or by a similar application to the skin, resulting in the characteristic tingling and feeling of numbness; together with its introduction into some small animal, hypodermically.

If the poisoning has occurred from swallowing the leaves or root of the plant, a careful microscopic inspection of the matters vomited and purged should be made, in order to identify its botanical characters.

In 1844, a trial took place in Albany County, New York (State *v.* Hendrickson), for alleged poisoning by aconite. It is chiefly remarkable for the testimony of two of the State's "experts," in relation to the methods of identifying this poison; and it furnishes another instance of the folly of attempting to diagnosticate a case of poisoning by such a substance, by a partial and incomplete mode of procedure. For instance, the medical man who made the post-mortem examination *inferred*, "from the emptiness of the stomach and small intestine, the corrugation of their mucous coat, and the presence of a reddish, viscid mucus in the stomach, *that vomiting had taken place*, and *that this vomiting was produced by aconite*"*!* The individual who undertook the chemical analysis testified "that he tested *a small portion of the stomach and a small portion of the duodenum* for prussic acid, and for most of the mineral poisons; then for morphine, strychnia, 'stramonine,' and other poisons, none of which he discovered." He then inferred the presence of *aconitine* from the fact that, after digesting a small portion of the stomach and duodenum in alcohol, evaporating, filtering, and purifying finally with animal charcoal, and then testing the filtered solution by boiling in sulphuric acid, it was "*turned to a deep port-wine red* color"! (See review of this trial, by Prof. C. A. Lee, in Am. Jour. Med. Sci., Oct., 1844.)

Detection in organic mixtures.—Aconitia may be recovered from organic mixtures and from the contents of the stomach by means of Stas' process, somewhat modified, as described for *nicotina* (*supra*, p. 462). Chloroform is preferable to ether, as the ultimate solvent for depositing the alkaloid. The residue thus obtained should be dissolved in a few drops of distilled water very slightly acidulated with acetic acid, and a drop of the solution should first be applied to the tongue, in order to recognize the peculiar tingling impression caused by aconite. Without a distinct recognition of this physiological proof (a large quantity of the solution being used for this purpose, if necessary, and the experiment repeated), it will be very unsafe to rely upon any mere chemical reaction, for the reason already mentioned. If, however, the physiological evidence is decided, the solution should be subjected to all the reactions mentioned above.

Dr. Wormley (*loc. cit.*, p. 620) states that by means of the process described, he was able to establish the presence of aconitia in the stomach of a dog, which had been killed in fourteen minutes by a drachm of the ordinary tincture. He also succeeded in detecting aconitia in the *blood* of a small dog, poisoned with forty minims of the tincture of the root. The animal died in sixty-four minutes after swallowing it. Twelve fluidrachms of its blood were treated as above; the ultimate chloroform deposit was stirred with two drops of water containing a trace of acetic acid: a drop of this placed upon the tongue gave positive evidence of the presence of the alkaloid. The remaining drop, diluted with two drops of pure water, was examined by the carbazotic acid, the chloride of gold, and the bromine test, with satisfactory results. It was estimated that in this instance not more than one three-hundredth of a grain had been operated upon by the various tests.

SECTION IV.

POISONING BY CALABAR BEAN (PHYSOSTIGMA VENENOSUM).

The *ordeal bean of Calabar* is a large, leguminous seed, from an inch to an inch and a half long, and of a brownish-black color. It is imported from the west coast of Africa, where it is used by the natives as the ordeal test for witchcraft,—the suspected person being compelled to drink a decoction of the poisonous beans. It owes its activity and poisonous properties to the alkaloid *physostigmia*, also named *eserina:* this resides chiefly, if not exclusively, in the cotyledons. These, when touched with nitric acid, assume an orange tint; and a yellowish brown when treated with perchloride of iron (Guy).

Effects on the system.—We are indebted chiefly to the researches of Dr. Fraser, of Edinburgh, for our knowledge of the action of physostigmia on the animal economy. (See Trans. Roy. Soc. Edinb., vol. xxiv.) A small, fatal dose given to one of the lower animals first occasions a slight tremor, extending from the hind quarters to the fore limbs and head, and then paralysis and muscular flaccidity, setting in in the same order. The rectum and bladder are then emptied. The pupils generally contract; the breathing becomes slow, irregular, and stertorous, and a frothy mucus escapes from the mouth. There appears to be a complete abolition of all reflex action, although sensibility is evinced whenever the animal is injured in any way, so long as the condition of the motor system allows it. This is exactly the opposite of the action of strychnia, which, as we have seen, so remarkably increases the excito-motor power. The muscular tremors persist throughout the paralysis; and they sometimes even assume the force of real convulsions (Fraser). Consciousness is preserved to the last, the animal dying quickly, as the respirations become progressively fainter. The pupils usually dilate immediately after death. The heart is found to be still beating for some time after death, unless the animal has perished from the effects of a very large dose of the poison; in which case its paralyzing force appears to have extended to this organ in common with the other muscles of the body. But even here the contractile power of the heart will respond to the galvanic current.

On *man* the symptoms are similar in character to those described in the preceding paragraph. There are giddiness, great muscular weakness, a sense of general torpor, and of faintiness; feebleness of heart-action, with, generally, contraction of the pupils, and occasional vomiting and purging. The consciousness is preserved. (See Phar. Jour., 1855, p. 474, for a full description of the effect of Calabar bean, as experienced by himself, by Sir R. Christison.)

The physiological action of physostigmia is precisely the reverse of that of nux vomica and strychnia: it appears to be a *direct spinal depressant*, while the latter is a true *spinal excitant.* For this reason it has been used beneficially in the treatment of *tetanus*, and likewise in the tetanic convulsions of *strychnia-poisoning.*

Its most characteristic physiological action is the property of contracting the pupil,—which serves at once to distinguish it from belladonna and the other mydriatics (see *ante*, p. 436). It differs from conia and woorara (which it resembles in some of its effects) in causing contraction of the pupils.

Treatment.—Only a few cases of poisoning by Calabar bean, in the human subject, have been reported. In such cases free emesis should be at once practiced, and the cautious administration of *atropia* be employed hypodermically, commencing with about the thirtieth of a grain, and gradually increasing it until dilatation of the pupils is manifested. From the carefully-conducted experiments of Dr. Fraser (*loc. cit.*) and others, there undoubtedly exists a real antagonism between these two powerful poisons, so that they may justly be regarded as being mutually antidotal.

Chemical properties.—*Physostigmia* is a colorless alkaloid, crystallizing in thin rhomboidal plates, and having a slightly bitter taste. It is sparingly soluble in water, but much more so in alcohol, chloroform, and ether. It forms salts with the acids. A water solution of it, and of its salts, in contact with potassa or soda, when exposed to the air, acquires a red color (in consequence of the absorption of oxygen), which subsequently changes to yellow, green, or blue. This property is said to detect the one-hundred-thousandth part of the alkaloid in solution. *Bromine in bromide of potassium* (the most delicate test for atropia) will precipitate a very dilute solution of physostigmia. Dragendorff found it to act in a solution of one ten-thousandth part. It gives a red color with less than one-thousandth of a grain. The *chloriodide of potassium and mercury* also precipitates it in a very dilute solution. *Chloride of gold* throws it down as a blue precipitate, from which the gold soon becomes reduced. According to Dr. J. B. Edwards (Med. Times and Gazette, 1864), it reacts with sulphuric acid and bichromate of potassa very much like strychnia,—yielding a violet color, which passes into red.

The most satisfactory test is probably the *physiological* one. This consists in placing a drop or two of the suspected poison into the eye of a rabbit, or other small animal: contraction of the pupil will take place in the course of fifteen or twenty minutes if physostigmia be present. Dragendorff has succeeded in separating it from the tissues by a process similar to that of Stas, employing benzole instead of ether as the solvent. It is said to be rapidly eliminated from the body by the saliva and the excretions under putrefaction. (Husemann's Jahresbericht, 1872, p. 570.)

CHAPTER XXVII.

ASTHENICS.

This subdivision of Cerebro-Spinants comprises those poisons which destroy life by *asthenia*, or failure of the heart's action. It is not intended to assert that death may not be produced by them, in some cases, in another way, as *e.g.* by shock, or by asphyxia. But, as their most strongly-marked symptoms are such as indicate a failure of the action of the heart, this name answers sufficiently well for grouping together a few of the neurotic poisons that especially display this property. The two most important members of this group are Hydrocyanic Acid and Digitalis. Cocculus Indicus is considered under the same head, merely for the sake of convenience.

SECTION I.

POISONING BY HYDROCYANIC ACID.—PRUSSIC ACID.

Hydrocyanic acid is one of the most energetic and rapidly-fatal poisons known. According to the statistics, its employment for criminal purposes is on the increase both in Europe and in this country. It occurs as a natural product in various vegetables, as the bitter almond, the kernel of the peach, apricot, plum, and cherry, the pips of apples, and the flowers of the peach and cherry-laurel. From the latter, a very poisonous water is distilled (cherry-laurel-water). It also exists in the root of the mountain ash. Properly speaking, hydrocyanic acid does not pre-exist in these vegetable substances, but is the product of their decomposition by the reaction of water, at a certain temperature.

Prussic acid, in its pure state (anhydrous), is a compound of cyanogen and hydrogen, HCy. It is a colorless, limpid liquid, extremely volatile, and having the odor of bitter almonds. It is among the most powerful and rapidly-fatal poisons known: a single drop placed upon the tongue of a large dog caused death in a few seconds. The anhydrous acid is very rarely met with except in the laboratory of the chemist: it possesses no medico-legal interest. It is the dilute or *medicinal* acid that is so frequently the cause of death.

The dilute or medicinal acid is merely a solution of the anhydrous acid in water. It occurs in the shops under two forms: (1) *the officinal acid*,—of the average strength of two per cent.; and (2) *Scheele's acid*,—of the average strength of five per cent. It should, however, be remarked that specimens of both varieties of the dilute acid vary considerably in strength; some samples of the commercial article being found not to contain a trace of the acid. This may be accounted for, at least partially, by the proneness to decomposition of the dilute acid when exposed to the action of light and air. The dilute acid is colorless, and has the bitter-almond odor, and a hot, pungent taste.

Symptoms.—These vary with the size of the dose. If taken in a large quantity—half an ounce to an ounce of the dilute acid—the symptoms usually commence in the act of swallowing, or in the course of a few seconds. It is seldom that they are delayed beyond one or two minutes. Tardieu describes them as coming on with lightning rapidity. There is an immediate loss of muscular power; the patient staggers, and falls to the ground; the respiration becomes hurried and gasping, the pulse imperceptible, the extremities cold, the eyes glassy and prominent, the pupils dilated and insensible to light; and sometimes convulsions occur. Towards the end, the respiratory movements appear to be suspended for a time, and then to be performed in convulsive fits, like sobbing, with forcible expiration. Occasionally the bladder and rectum are evacuated involuntarily. As regards the occurrence of a peculiar *cry*,—such as is frequently heard in animals poisoned by prussic acid,—the experience of all observers is against its existence in the human subject. A strong characteristic odor of bitter almonds is usually exhaled from the patient. The face is either livid or pallid; the jaws are spasmodically closed; there is frothing at the mouth; and death occurs sometimes in a violent convulsion, and at other times is preceded by coma, with stertorous breathing. This latter symptom (stertorous breathing) should not be overlooked, as it might possibly lead to a mistake in the diagnosis. It is particularly alluded to by Christison, Taylor, and Tardieu.

Fatal period.—Death generally occurs within ten to fifteen minutes after swallowing a fatal dose of the poison. Rarely is it protracted beyond half an hour. One case is recorded where one hour supervened. Although the fatal result is so speedy, the insensibility is not always immediate. This is a circumstance of some medico-legal interest, as where, in a doubtful case, the suspicion of suicide might seem to be negatived by the fact of the deceased being found lying calmly in bed, the bedclothes properly adjusted, and the vial that contained the poison corked, and put away in a bureau-drawer, or in a distant part of the room. From numerous cases on record, there is no doubt that, after swallowing even a large dose of prussic acid, a sufficient length of time is allowed to perform all the above, and other voluntary acts, before the fatal insensibility sets in. One or two instances may be cited to sustain this assertion. Dr. Sewall reports (Boston Med. and Surg. Jour., xxxvii. p. 322) the case of a gentleman who, after swallowing seven drachms of Scheele's acid, equivalent to twenty-one grains of the anhydrous acid, walked from the table in the middle of his room to the door, unlocked it, called for assistance, then walked to a sofa, and stretched himself upon it; a little while afterwards he was found in an insensible condition, with stertorous breathing, and he soon died. Dr. Taylor mentions another remarkable case—that of a gentleman, aged forty-four, who swallowed half an ounce of prussic acid (strength not stated). He then walked ten paces to a flight of stairs, descended the stairs, seventeen in number, and went to a druggist's shop forty-five paces distant, where he had previously obtained the poison, entered the shop, and said, in his usual tone of voice, "I want some more of that prussic acid." He then became insensible, and died in from five to ten minutes after taking the poison, without convulsions.

When taken in a large though non-fatal dose, the symptoms are confusion of head, giddiness, a sense of pressure on the brain, great loss of muscular power, inability to stand, nausea, and occasional vomiting, foaming at the mouth, and tetanic convulsions. This latter symptom is, however, more apt to be the result in fatal cases. The respiration is always difficult and oppressed; and several days may elapse before the health is completely restored.

When applied externally to the skin, this poison has produced serious and even fatal consequences. M. Tardieu (Sur l'Empoisonnement, p. 1034) relates the case of a photographer, who, wishing to remove some stains of nitrate of silver from his hands, rubbed them over with a piece of cyanide of potassium, and inadvertently got a fragment under one of the nails. He shortly experienced a sharp pain, which was followed by extreme vertigo. In order to rid himself of the offending substance, he unfortunately applied vinegar to his hand, which had the effect of decomposing the cyanide and liberating free prussic acid. The vertigo was greatly increased, accompanied with rigors, pallor of the face, excessive loss of muscular power, and inability to speak, but without loss of consciousness. This condition lasted for nearly ten hours.

The same author mentions the case of a medical student, who very nearly lost his life from incautiously breathing the vapor of prussic acid which escaped from a vessel in which he was preparing it.

Sir R. Christison reports the case of a man in whom the liquid acid applied to a wound in the hand caused death in an hour afterwards.

Fatal quantity.—From numerous cases reported, we may conclude that nine-tenths of a grain of anhydrous acid, equivalent to about fifty minims of the usual medicinal acid of the shops, is the smallest fatal dose for an adult. Mr. Hicks reports a case of this kind (Med. Gaz., vol. xxxv. p. 896).

The largest dose from which an adult recovered was probably in the case reported by Mr. Burnam (Lancet, Jan. 14, 1854), in which one drachm of Scheele's acid, equivalent to 2.4 grains of anhydrous acid, was swallowed by mistake. The individual recovered, most probably because remedies were immediately applied (inhalation of ammonia and the cold affusion).

Sir R. Christison has reported a case (Brit. and For. Med.-Chir. Rev., April, 1854) in which a gentleman who had taken a little less than two grains of the anhydrous acid was with great difficulty recovered. Mr. Bishop relates an instance (Lancet, Sept., 1845, p. 315) of a man who entirely recovered after taking forty minims of a solution containing one grain and a third of anhydrous acid. In both these cases, as in the former one, the recovery was undoubtedly owing to the prompt and vigorous measures adopted.

Treatment.—Such is the rapidity with which prussic acid produces its fatal effects, that there is scarcely any opportunity for the employment of remedies. The *cold affusion*, consisting in the dashing of cold water over the face and chest of the patient, has been found, on the whole, the most efficient remedy. The cautious inhalation of ammonia and chlorine vapors may also be employed, along with stimulants internally and externally applied. As a *chemical antidote*, it has been proposed to use a mixture of the protosulphate and sesquisulphate of iron, followed by a solution of carbonate of potassa. Such a mixture would produce with prussic acid, if present in the stomach, the insoluble and inert *Prussian blue.* This experiment has been found successful in animals.

Post-mortem appearances.—The body is said to maintain its rigidity longer than usual. Its putrefaction is neither hastened nor retarded. The face is either pallid or livid; the eyes are often glistening and staring, with the pupils dilated; the lips blue; jaws firmly set, with, at times, a bloody froth about the mouth. The blood throughout the body is fluid, and of a dark-blue color. The vessels of the brain are congested. Tardieu (*loc. cit.*, p. 1037) speaks of sanguine and sero-sanguine effusions at the base of the brain as an occasional occurrence, and one that might lead to suspicion of apoplexy, which, however, would be cleared up by the absence of hemiplegia, and by the rapidity of the death.

The lungs and liver are congested; and the mucous membrane of the stomach, especially about the cardiac extremity, is described as being nearly always in a state of high congestion.

The exhalation of the peculiar *odor* of the acid is one of the most important post-mortem characters. This odor is sometimes perceived even before the body is opened, especially in recent cases, but is particularly noticeable on opening the cavities of the abdomen and thorax, often when the brain is opened, and more frequently in cutting into the stomach. But, as the poison is extremely volatile, it may happen that the odor will have completely disappeared in a few hours, or days, after death, especially if the body has been much exposed. Again, the odor may be disguised by other more powerful smells, as of tobacco, mint, etc., or concealed by the putrefactive odor. The mere absence of the characteristic odor is, therefore, by no means a proof of the non-existence of the poison.

In the case of the seven Parisian epileptics, who died in periods varying from fifteen to forty-five minutes, no odor of the poison was perceived in any part of the body twenty-four hours after death, although the dose was a large one—over five grains of anhydrous acid (Braithwaite's Retrospect, xii. p. 125). On the other hand, in Mr. Hicks's case, in which only nine-tenths of a grain of the acid were taken, the odor of the poison was plainly perceived on opening the chest, and was also strongly emitted from the contents of the stomach, *ninety hours* after death.

Chemical analysis.—There are *four* recognized tests for prussic acid, which may be briefly designated as the silver, iron, sulphur, and copper tests. The first three are characteristic; and they all may be applied to the acid either in its form of liquid, or vapor.

1. *The silver test.*—A solution of hydrocyanic acid, or of a soluble cyanide, gives with a solution of nitrate of silver a white, crystalline precipitate, distinguishable from the white

chloride, as follows: (1) by its crystalline character (prisms and needles): the chloride is amorphous; (2) its sparing solubility in ammonia: the chloride is readily soluble; (3) the permanence of its color when exposed to light: the chloride becomes dark-colored; (4) its solubility in boiling, strong nitric acid: the chloride is insoluble; (5) when perfectly dried and heated in a small reduction-tube, the cyanide is decomposed, giving off free cyanogen gas, which burns with a characteristic roseate flame. One-hundredth of a grain of the cyanide may thus be recognized.

A satisfactory method of identifying the cyanide of silver is the one proposed by MM. Henry, Jr., and H. Hubert, and recommended by Orfila and Tardieu. The supposed cyanide, after thorough washing and drying, is introduced into a small glass tube closed at one end, from five to seven inches long, and containing at its closed extremity a rather less quantity of pure iodine. On heating this end of the tube very gently, beautiful snow-white crystals of *iodide of cyanogen* are deposited upon the cool portions of the tube. These crystals may be preserved indefinitely in sealed tubes; and they may be used for the production of Prussian blue, by dissolving them in a solution of potash and adding a mixture of the protosalt and persalt of iron.

The silver test is particularly delicate when applied to prussic acid in the state of *vapor*. For this purpose, the material containing the poison (such as the stomach, etc., cut into pieces and diluted, if necessary, with distilled water) is put into a wide-mouthed flask, and a watch-glass containing a drop of nitrate of silver solution on its concave surface is inverted over the open mouth of the flask, which may be gently heated by immersion in warm water. The vapor of the acid rises, and, coming in contact with the nitrate of silver, forms an opaque, white spot—the cyanide of silver—which can easily be recognized by the microscope and the other tests mentioned above, besides being corroborated by the sulphur and iron tests, as will be shown presently. If, however, the material is in an advanced stage of putrefaction, this vapor test cannot be applied, since the black sulphide of silver, resulting from the sulphuretted hydrogen of decomposition, would obscure the white cyanide, even if present. The silver test, as thus employed, is the most delicate of all the vapor tests. According to Wormley (Micro-Chem. of Poisons, p. 179), one hundred-thousandth of a grain of prussic acid can thus be recognized. For this delicate manipulation, a single drop of water containing one hundred-thousandth of a grain of prussic acid is put into a watch-glass, over which is placed another glass holding on its concave surface a small drop of solution of nitrate of silver. On warming the lower glass by the hand, the vapor of the hydrocyanic acid will escape and act upon the silver solution, producing a whitish deposit, more evident at the margins of the drops. The crystals can be identified by the microscope. Prof. Guy (Forensic Medicine, p. 575) mentions that a single apple-pip, bruised and moistened with water, and placed in a watch-glass, yielded twenty-two distinct reactions, —each spot exhibiting by the microscope crystals of cyanide of silver.

2. *The iron test.*—This consists in adding to the suspected solution a little liquor potassæ, and then a mixed solution of the protosulphate and persulphate of iron: a dirty-greenish-blue precipitate is thrown down, which, on the addition of a few drops of pure hydrochloric acid, becomes the clear *Prussian blue.* If the amount of the hydrochloric acid be very small, the color of the solution, after adding the hydrochloric acid, will be greenish blue, and a considerable time may elapse before any really *blue precipitate* takes place. This last result, however, should always occur, if prussic acid has been present, even though in minute quantity; and time should be allowed for the precipitate to form, since this is permanent, and (in a criminal case) can be exhibited to the court and jury, as a characteristic evidence of the poison.

In the uncertainty arising from the presence of only a minute quantity of the poison, it is recommended to throw the disturbed liquid, after the addition of hydrochloric acid, upon a small white filter-paper: the yellowish liquid portion will thus be removed, and the blue deposit on the paper, after being washed with water slightly acidulated, will become very distinct upon the white ground of the paper. The latter, moreover, when dried, can be preserved and exhibited as evidence at the trial.

In manipulating with minute portions of prussic acid with the iron test, caution should be used in the proper adjustment of the reagents. Too much potash will redissolve the precipitated Prussian blue, and an excess of iron solution may likewise retain it in solution.

The iron test may also be used as a *vapor test.* Moisten the watch-glass with a drop of liquor potassæ, and, after exposure to the suspected vapors, add a drop or two of the mixed solution of protosulphate and persulphate of iron, and develop the Prussian blue by a drop of dilute hydrochloric acid.

3. *The sulphur test* (Liebig's test).—If sulphide of ammonium be added to a solution of hydrocyanic acid, and gently heated to dryness, a white sulphocyanide of ammonium is formed: when this is touched with a drop of perchloride or persulphate of iron, a beautiful blood-red sulphocyanide of iron results, which is very characteristic and conclusive of the presence of prussic acid, in the absence of meconic acid and the soluble acetates. (The meconate and the acetate of iron both possess a reddish color, but they can easily be identified. See *ante*, p. 377.)

The sulphur test is very advantageously employed as a vapor test, as follows. Moisten a watch-glass with a drop of sulphide of ammonium, and invert it over the vessel containing the prussic acid; on gently heating the vessel, the vapor of the acid will rise, and form the sulphocyanide of ammonium upon the watch-glass. When this is allowed to dry, by evaporation, and a drop of the neutral persalt of iron is applied to it, the blood-red color is immediately developed. If the evaporation be not complete, the application of the iron salt may produce a black stain (sulphide of iron), which may obscure the result.

The sulphur test may also be applied to confirm the silver test. For this purpose, the spot of cyanide of silver on the watch-glass should be moistened with a drop of sulphide of ammonium, and, when thoroughly dried, touched with a drop of the persalt of iron: the characteristic blood-red color may be seen, in spite of the black sulphide with which it is associated.

4. *The copper test.*—The liquid is first rendered slightly alkaline by liquor potassæ, and, on adding a dilute solution of sulphate of copper, a greenish-white precipitate is thrown down: on the addition of a little hydrochloric acid, the precipitate becomes nearly white.

This test may also be used in the form of vapor, the watch-glass being moistened with the copper solution, made slightly alkaline, and, after exposure, a drop of dilute hydrochloric acid being added.

As regards the relative delicacy of the tests described, experiments show that for the *liquid* hydrocyanic acid the iron and the sulphur tests both exceed the silver test; but for the acid in the form of *vapor*, the silver test far surpasses all the others.

A new test, and one of extreme delicacy, which is attributed to Schoenbein, has lately been brought to notice. It is made as follows. Dissolve forty-five grains of guaiacum in three ounces of alcohol, and with this solution saturate a sheet of thin, white filter-paper, which is then to be gently dried, and cut into proper slips. Next dissolve fifteen grains of sulphate of copper in an ounce and a half of distilled water. When the test is to be applied, dip a slip of the test-paper into the copper solution, and hold it over the vessel or substance containing the hydrocyanic acid; very soon the paper assumes a deep-blue color. The author of the test states that it will detect one-millionth of a grain of the acid. (Brit. and For. Med.-Chir. Rev., Oct., 1869.)

This is certainly a test of extraordinary delicacy, as we have ourselves verified by actual experiment. A single drop of ordinary medicinal prussic acid (two per cent.), when diluted in an ounce of water, will readily produce the blue color upon the test-paper. This is about equivalent to one part of anhydrous acid diluted with nearly twenty-nine thousand parts of water. Unfortunately, this test is not *characteristic* of prussic acid, since the same blue color is brought out by the presence of *ozone* in different forms. Besides, in experimenting in cases where the hydrocyanic acid was supposed to be present in exceedingly minute quantity, and the vessel believed to contain it was warmed, we have found that the mere drying of the test-paper will cause it to assume a blue color *even in the absence of the poison.*

Process by distillation.—This process is usually resorted to for the purpose of separating prussic acid from organic mixtures, as the stomach, blood, articles of food, etc. The mixture should first be carefully examined for its odor, and then tested for the presence of *free* prussic acid. If not found to be distinctly alkaline, it should be distilled *without* the addition of any sulphuric acid. It is very important in a toxicological investigation to remember this latter point. Supposing no free hydrocyanic acid to be present in the contents of a stomach, and these to be subjected to distillation along with sulphuric acid, it might readily happen that the sulphocyanide of potassium that exists in the saliva as a normal constituent (and which, of course, would get into the stomach), as well as any ferrocyanide and ferricyanide of potassium which might accidentally be present, would undergo decomposition in the act of distillation, and traces of the *developed* prussic acid would be discovered by the usual tests. We therefore consider it an error, in a medico-legal case, to employ any acid along with the suspected materials in the process of distillation, since by so doing the chemist inevitably puts it out of his power to determine whether the traces of prussic acid which he discovers are due to the poison originally existing in the free state, or merely to the prussic acid which he has actually *manufactured* by the process used. Of course, if cyanide of potassium had been the poison employed, the contents of the stomach would have an alkaline reaction; in which case the addition of sulphuric or some other acid would be perfectly proper.

In employing the distilling process, the materials should, if solid, be cut into small fragments, and a sufficient quantity of distilled water added; they should then be introduced into a proper-sized glass retort, connected with a good condensing arrangement. The retort should not be heated beyond 200° F. About one-fourth of the contents should be allowed to distill once: these should be subjected to the different tests already mentioned. They frequently reveal the characteristic odor.

In the celebrated case of Dr. Paul Schoeppe, at Carlisle, Pa., in 1868 and 1872, where the allegation first was, that the deceased had been poisoned with prussic acid, and subsequently that it was by a mixture of prussic acid and morphia, the defense very properly took the ground that the "faint traces" of prussic acid alleged to have been discovered by the analyst were really produced or *manufactured* by the process which was employed for its detection, viz., *distillation with sulphuric acid;* and for the reason mentioned above—the decomposition of the sulphocyanide of the saliva by the acid. This view seemed, moreover, to receive confirmation from the fact that the *traces* described by the analyst must have been exceedingly *faint*, inasmuch as the iron test merely revealed what was described to be a bluish discoloration, *which did not become a definite precipitate:* the actual precipitate is always essential for proof. The sulphur test (the only other one employed) yielded a rather equivocal reddish coloration. Moreover, there was an entire absence of any characteristic symptom of the alleged poison, before death (see *ante*, p. 93).

One of the latest American authorities, speaking of this acid, says: "It may certainly become a question of serious import whether the traces of it found afterwards may not be due to some other cause than its ingestion into the stomach. Thus, if the contents of the stomach be subjected to distillation *with an acid*, it may possibly happen that the sulphocyanide of potassium, which sometimes exists in minute traces in the saliva, may be decomposed, and evidences of prussic acid be thus obtained." (Wharton and Stillé's Med. Jurisp., 1873, ii. p. 515.)

The source of the poison found in the distillate, when an acid has been employed, may be determined by treating a portion of the reserved liquid with a few drops of hydrochloric acid, stirring the mixture for a short time, and then adding a solution of the perchloride of iron. If the liquid treated contains either a ferrocyanide or a sulphocyanide, the former will be indicated by the formation of Prussian blue, and the latter by the production of the red sulphocyanide of iron; whereas a simple cyanide (cyanide of potassium) will not give any reaction under the circumstances. As commercial cyanide of potassium may be contaminated with the ferrocyanide, traces of the latter may be present in poisoning by the former.

As regards the question whether prussic acid may be *spontaneously* generated by the distillation of putrescent animal matters, Orfila appears to have inclined to this belief, and has recorded some experiments that seem to countenance it (Toxicologie, ii. p. 465). Later authorities, however, discredit it. Still, we are of the opinion that in an important medico-legal case, involving the life of the accused, something more should be insisted upon as proof than the finding of *mere traces* of prussic acid, since these might possibly be the result of some spontaneous animal decomposition, brought about under conditions not yet perfectly understood. Especially should this be insisted upon where the symptoms preceding death did not agree with those characteristic of the alleged poison.

Period after death when the poison may be found.—On account of its great volatility and its ready decomposition, all traces of prussic acid may disappear very shortly after death. Prof. Casper mentions a case in which it could not be discovered twenty-six hours after death. On the other hand, Dr. Taylor alludes to two cases in which the poison was identified respectively in twelve and seventeen days after death.

Mr. West was able to detect it on distillation by the odor and by the silver and iron tests *twenty-three days* after death, although no pains had been taken to insure its preservation (Prov. Med. Jour., July 23, 1845); and M. Brame was equally successful, after the lapse of a similar period, in the case of a young man of Tours, who had poisoned himself with a very large dose of prussic acid (Comptes-Rendus, No. 20, Nov. 13, 1854). In a German case it was also detected three weeks after death (Brit. and For. Med.-Chir. Rev., April, 1860).

The mere fact of *putrefaction* is no obstacle to its detection; but when the viscera containing the poison have undergone putrefaction, no traces of it may be discoverable either by its vapor or by distillation. In this case it may have been converted into sulphocyanide of ammonium by the sulphide of ammonium resulting from the putrefaction. The sulphocyanide should be dissolved out of the dried viscera or liquids by alcohol, and the solution evaporated to dryness; the residue is then dissolved in water, and tested by a persalt of iron.

Quantitative analysis.—The free hydrocyanic acid is precipitated by nitrate of silver, as a cyanide; this, when washed and dried, is weighed: every 100 parts correspond to 20.15 parts of anhydrous acid.

CYANIDE OF POTASSIUM.—This substance is now extensively used in the arts, especially in the processes of photography and electrotyping. It is exceedingly poisonous, causing death in doses under five grains.

It is a white, crystalline, deliquescent salt, very soluble in water; its solution, when pure, is colorless; giving off the strong odor of prussic acid; it has an alkaline reaction. It is not very soluble in alcohol. The *symptoms*, *post-mortem appearances*, and *treatment* are similar to those of prussic acid.

Chemical analysis.—1. It is decomposed by all acids, setting prussic acid free, which may be identified by the usual tests. 2. It gives, with a solution of nitrate of silver, the white cyanide. 3. The potash is precipitated by tartaric acid and bichloride of platinum. 4. The iron and copper tests, as applied to prussic acid, act similarly here, only the addition of liquor potassæ is not needed.

In organic mixtures, the prussic acid may be obtained by neutralizing the alkali with sulphuric acid, and distilling it at a low temperature.

OIL OF BITTER ALMONDS.—This is procured by the distillation of the pulp or emulsion of bitter almonds. It contains a variable proportion—amounting to from eight to fourteen per cent.—of anhydrous prussic acid, together with hydride of benzule, benzoin, and benzoic acid.

Its poisonous properties are due to the prussic acid. When entirely freed from the latter, the oil is stated to be harmless.

Properties.—Ordinary oil of bitter almonds has a light-yellow color, a peculiar, pungent odor, due to the prussic acid, and a bitter, pungent, and aromatic taste. It is heavier than water, which only partially dissolves it; it is soluble in alcohol and ether; it has a slight acid reaction. A liquid sold as *almond flavor*, or *essence of peach-kernels*, consists of this oil dissolved in seven or eight parts of spirit: it is too dangerous a substance for domestic use.

Oil of bitter almonds is a violent poison, producing the same effects as prussic acid. The *symptoms*, *post-mortem appearances*, and *mode of treatment* are the same as those already described under the head of prussic acid.

The *fatal dose* is about twenty drops.

Cherry-laurel-water, obtained by distilling the leaves of the cherry-laurel (*Prunus lauro-cerasus*), contains also a portion of an essential oil similar to the oil of bitter almonds. It owes its poisonous properties to the hydrocyanic acid it contains. Cherry-laurel-water has more than once proved fatal; but it has been specially identified with the celebrated case of Sir Theodosius Boughton, who was poisoned by his brother-in-law, Captain Donallan, in 1781.

The kernels of the *apricot*, *peach*, and *cherry* have all proved poisonous—in some instances fatally so—when swallowed by children. Dr. Keating has reported (Trans. of Phila. Coll. of Physicians, vol. iii. No. 3) an interesting case, in which he succeeded, by the affusion of cold water, in restoring a child three years of age, who had eaten a quantity of peach-kernels. The child was seized suddenly, and, when seen, was insensible, with slow, deep, sobbing respiration, no convulsions of the limbs, but slight twitching of the mouth, cold extremities, finger-nails livid, hands tightly clinched, eyes prominent, and pupils dilated. A strong odor of prussic acid was perceived about the mouth. An emetic brought up a quantity of peach-kernels, emitting the characteristic odor.

The case is supposable in which death is alleged to have resulted from prussic acid, and where the chemical analysis has revealed *traces* of this poison, that these should be ascribed, by the defense, to kernels of the peach or apricot, or even to apple-pips, found in the stomach of the deceased. An instance of the latter is mentioned by Taylor (On Poisons, p. 602). Any doubts in the matter would be cleared up by finding a larger quantity of hydrocyanic acid than could be satisfactorily accounted for from the above substances. Moreover, death could hardly result from the ingestion of these, without their subsequent discovery in such large quantities as would entirely preclude the idea of the administration of prussic acid in substance.

NITRO-BENZOLE, OR ESSENCE OF MIRBANE.—This substance is a product of the action of nitrous acid on benzole. It is a pale, lemon-colored liquid, with a strong odor resembling that of bitter almonds. It has of late years been introduced into use in perfumery and confectionery as a cheap substitute for the oil of bitter almonds. It is a powerful narcotic poison, resembling in its general effects those of the oil of bitter almonds or prussic acid, although much slower in its operation than the two latter. After the first characteristic symptoms have continued for about four hours, the patient falls suddenly into a coma as in an apoplexy, which usually proves fatal in about five hours.

In a fatal case described by Dr. A. Taylor (Med. Jurisp., p. 310), the appearances after death were—flushed face, livid lips; the superficial vessels of the body, especially about the throat and arms, were gorged with black and fluid blood; the lungs were somewhat congested. The cavities of the heart were full of blood; the liver was of a purple color, and the gall-bladder distended with bile; the brain and its membranes were congested, with much bloody serosity in the ventricles. Nitro-benzole, as well as aniline, into which it appears to be partially converted in the body, was detected in the brain and in the stomach.

This poison operates more powerfully in the form of vapor, than as a liquid: a number of fatal cases resulting from the inhalation of the fumes have been recorded. The rapidly-fatal cases might possibly be mistaken for apoplexy; but the poison would be identified by its powerful odor.

Chemical analysis.—It is distinguished from all other liquids, except oil of bitter almonds, by its odor; and from this oil by the following test. Pour a few drops of each upon a plate, and add a drop of strong sulphuric acid: the oil of bitter almonds acquires a rich crimson color with a yellow border, while the nitro-benzole produces no color. It gives none of the reactions of prussic acid with the ordinary tests of this acid.

When associated with organic liquids, it may be separated by first adding sulphuric acid, and then distilling.

SECTION II.

POISONING BY DIGITALIS (FOXGLOVE).—DIGITALINE.

The purple foxglove (Digitalis purpurea) is indigenous to Europe, and is cultivated as an ornamental plant in our gardens. All parts contain the poisonous principle *digitaline*, which, however, abounds most in the leaves of the second year's growth. According to most authorities, the fresh leaves contain less than one per cent. of the active principle; while Ch. Blaquart asserts that ten to twelve per cent. of *crystallizable* digitaline can be extracted from the crude drug (L'Union Pharmaceutique, Nov., 1872).

Symptoms and effects.—Cases of poisoning by digitalis are comparatively rare. Most of our knowledge of its toxic effects is derived from experiments on animals. Its chief and important impression is made directly upon the circulation: under its influence, the pulsations of the heart are diminished in frequency, but increased in power. Hence it is now generally regarded as a direct heart-stimulant. The poisonous symptoms, in man, are vomiting, purging, and severe abdominal pains; a sense of heat in the head, vertigo, and disordered vision; dilated pupils; the pulse may be full and slow in the horizontal position, but becomes feeble and rapid on the patient's sitting up. Prostration then comes on, with tendency to syncope; the pulse becomes feeble, small, and irregular, although the heart-beat may be strong and hard. The eyes are very prominent; the pupils fixed and dilated; the sclerotic, according to Tardieu, acquires a peculiar blue color, which he regards as an almost characteristic sign. Sometimes there is abundant salivation; the urine is generally suppressed. Towards the close there are usually delirium and stupor; and convulsions are very apt to precede death, which does not, as a rule, occur within twenty-four hours, and is sometimes postponed for several days. Tardieu mentions a case communicated by M. Barth to the *Société Anatomique*, 1849, of an anasarcous woman, who swallowed twenty-five grammes of tincture of digitalis that had been prescribed for external use. Death occurred in *three-quarters of an hour;* the only symptoms being copious vomiting, a general malaise, and a very severe abdominal pain (*loc. cit.*, p. 636). In cases of recovery, the patient is not fully restored to health for a considerable time,—sometimes for weeks.

The differential symptom of poisoning by digitalis is the irregular, intermittent, enfeebled pulse, which varies so remarkably between the supine and the erect posture: this, conjoined with the sense of heat and pain in the head, the violent vomiting and abdominal pains, the profound debility, and the troubled vision, will usually be sufficient to indicate the cause. Another very characteristic effect of digitalis is its *cumulative* power—its tendency suddenly to break out with extreme violence, after a continued apparent inertness.

The *post-mortem appearances* are turgescence of the vessels of the brain and redness of the lining membrane of the stomach.

The minimum fatal dose of either digitalis or its active principle is not known. A drachm of the *powder* has been taken without producing death; although most violent vomiting resulted. The *tincture* has been given in as large quantity as half a fluidounce without any serious result, although in medical practice the ordinary dose is from ten to thirty drops. Of digitaline, it is probable that one-fourth to one-half a grain might prove fatal. Orfila states that from one to two grains killed dogs in a few hours, unless speedily thrown off by vomiting.

DIGITALINE.—As this active principle possesses neither acid nor alkaline properties, it is classed among the *neutral* bodies. As usually met with, it is an amorphous powder, of a pale-yellowish color. It has, however, lately been obtained in the form of fine, white, needle-shaped crystals, by M. Nativelle (Pharm. Jour., 1872, April 27, p. 865). This crystalline substance is now generally admitted to possess about equal strength with the amorphous variety; but it is difficult to reconcile this with their alleged respective percentage in the crude drug. Thus, according to Taylor, Guy, and Pereira, the proportion of digitaline (amorphous) is only one per cent., while, according to Blaquart (as already noticed), that of the *crystallizable* variety is ten to twelve per cent.

There would seem to be a true physiological *antagonism* between digitaline and aconitia. According to Boehm (Pflüger's Archiv, Feb., 1872), in digitalis-poisoning of the frog, even when the heart has ceased to contract, its movements are restored by aconitia, muscaria, and delphinia; and Dobie reports a case (Brit. Med. Jour., Dec., 1872) of recovery after the ingestion of an ounce of Fleming's tincture of aconite, apparently due to the hypodermic injection of twenty minims of tincture of digitalis, and the exhibition by the mouth of three doses, within an hour, of a drachm (each) of the tincture, brandy, and ammonia (H. C. Wood's Therapeutics, p. 125). As yet, no case of digitalis-poisoning in man has been recorded where any of the above-named substances have been employed antidotally. It must be remembered that whilst there might exist a physiological antagonism, so far as their operation upon the heart is concerned, this might not extend to their influence upon the cerebro-spinal axis.

Chemical reactions.—As already stated, digitaline occurs under two forms—the amorphous and the crystalline. It has an intensely bitter taste. It is sparingly soluble in hot and in cold water; very soluble in alcohol, both cold and hot; almost insoluble in pure ether, but easily dissolved if the ether contains a little alcohol. Chloroform is one of its best solvents, and, according to MM. Homolle and Quevenne, is the one best adapted for its separation. Its aqueous or alcoholic solution gives no reaction with litmus or turmeric-paper, indicating its neutral character. The alkalies, even when diluted, gradually destroy its bitter taste. Cold, concentrated sulphuric acid imparts to it, at first, a brownish-black color, which gradually passes into a red. If it be warmed, the color rapidly becomes brown. If to the cold, brown sulphuric acid solution two or three times its volume of distilled water be added, it assumes a green color, and deposits a green powder, and the liquid gradually assumes a yellowish tint (Tardieu). Acetic acid dissolves it without color; strong nitric acid acts upon it energetically, with the escape of orange-colored fumes, imparting to it an orange-yellow color, which becomes a pale yellow on standing. Hydrochloric acid imparts to it a light-greenish tint. According to Tardieu, if the digitaline is perfectly pure, it is not colored at all by this acid.

M. Grandeau states that if sulphuric acid be applied to a small deposit of digitaline, obtained by evaporation of a solution, it assumes a rose-color, which, on exposure to the vapor of bromine, becomes a violet; but M. Tardieu denies that this is at all characteristic, since he found that numerous substances, including many of the animal secretions, when thus treated with sulphuric acid and bromine vapor, will yield a violet color, transient, but well marked (*loc. cit.*, p. 655). Its solution is precipitated by tannic acid, but not by bichloride of mercury, nor by chloriodide of potassium and mercury, both of which act upon the true alkaloids. It is not affected by iodic acid.

Detection in organic mixtures, or in the contents of the stomach.—As before remarked, poisoning by digitalis is comparatively rare: the cases heretofore reported have been chiefly the result of accident, and the majority of them have not proved fatal. At least one instance is on record where death resulted from *digitaline* administered homicidally—the celebrated case of *De la Pommerais*, which occurred in France in 1863. All that need be said under the present head may be included under the search for this poison in the dead body.

In a suspected case, the first duty of the toxicologist will be to institute a very careful examination of the interior of the stomach and intestines for any fragments or powder of the leaves, in case the poison has been swallowed in this form; if it has been taken in the form of tincture, the interior of the stomach may present a greenish color, and may even emit a peculiar odor, which will be suggestive. If digitaline has been taken, as this is generally found in the form of granules, a close inspection of the digestive canal may reveal some little remnants of the latter not thoroughly dissolved. The vomited matters especially demand attention on this account (*vide ante*, p. 99). After these preliminary investigations, the organs, cut into small pieces, are put into a large glass flask containing pure alcohol at 95°. This is gently heated on a water-bath, and frequently stirred, to favor the solution. After digesting twenty-four hours, the contents of the flask are strained, and filtered through paper, the solids being repeatedly washed with strong alcohol. All the washings being united to the filtrate, it is again filtered, and concentrated by evaporation to the consistence of a soft extract: this may be used for experiments on animals. Another solution of this extract in strong alcohol, followed by filtration and a new evaporation, will eliminate an additional quantity of foreign matter, and the resulting purified extract may also be employed for physiological experimentation. It is not recommended to precipitate the solution of the ultimate extract by tannic acid, as it has been shown by Tardieu and Roussin that the matter thus thrown down does not represent the active principle in such cases.

The above authorities also distinctly assert the impossibility of determining the presence of digitaline, in a medico-legal case, either by the post-mortem signs or by the chemical analysis. They deem it essential in every such case to resort to the *physiological* test; and this step seems to be justified, first, by the circumstance of the inability to identify the poison with any degree of certainty by a chemical analysis, and secondly, by the fact that it produces a perfectly-recognized impression upon a particular organ, the heart.

As the result of numerous experiments with this agent upon animals, by various persons, it seems well established, on the one hand, that death takes place from a sudden cessation of the heart's action, and on the other, that the ventricles undergo a most decided and rapid rigidity at the moment of death. In dogs, this occurs almost immediately after the last ventricular systole (Tardieu); and in frogs, according to Pelican, the ventricle comes to a stop always in the state of strong contraction. If, then, there be introduced under the skin of the thigh or abdomen of a frog a portion of the ultimate extract obtained from the vomit or from the organs of a person poisoned by digitaline, it will be found that the heart-beats lose their regularity, and, after six minutes, fall to sixteen pulsations; after twenty minutes, to one-half this figure; and after twenty-five minutes, to one-third; in half an hour they cease completely. Such, moreover, is the irregularity of the heart's action, that, notwithstanding the remaining strength of its pulsations, this organ never completely empties itself of blood; and when finally it ceases to beat, the ventricle is contracted, and the auricle dilated.

In experimenting with the suspected material upon either dogs or frogs, it will always be proper to institute a rigid comparison between the results thus obtained and those procured from similar experiments made with digitaline itself upon similar animals.

Tardieu reports a number of cases of poisoning by digitaline taken accidentally and suicidally,—the majority of them not fatal. He also gives the medico-legal report of the celebrated case of *Couty de la Pommerais*, a homœopathic practitioner of France, who was tried and convicted for poisoning a woman named Pauw, with digitaline, in the year 1864. The circumstances connected with this unique case are of such a character as to justify our giving a short abstract in this place. The prisoner had renewed his intimacy with this woman, who was forty years of age, after a long absence, and had induced her to insure her life in various insurance-offices for a very large sum of money, quite disproportionate to her circumstances. Very soon after this, being previously in good health, she was suddenly seized with violent vomiting, and died after an illness of twenty-four hours. Immediately after her death, he put in a claim for the insurance-money. Suspicion was aroused, and the body was disinterred and examined thirteen days after death. The examiners, MM. Tardieu and Roussin, found all the organs perfectly healthy: they revealed no natural cause of death. A chemical analysis of the alimentary canal revealed no poison. The symptoms during life, so far as they were imperfectly recollected, were excessive vomiting, with great depression of the heart's action, and exhaustion. Failing to detect any poison by the chemical research, they resorted to the *physiological* test,—the administration of the extract, obtained from the stomach and bowels, to small animals, the effects of which were the exciting of repeated vomitings and a very notable diminution of the number of heart-beats: the action of the heart was irregular and intermittent, and the respiration was deep and painful.

Another experiment was made with an extract obtained from the scrapings of the floor *upon which the deceased woman had vomited:* it was introduced into the thigh of a dog, and the animal, after suffering from vomiting and depression of the heart's action, died in twenty-two hours, without coma or insensibility. Thirty-one grains of the same extract administered to a rabbit proved fatal in less than three hours. This extract possessed all the chemical properties of digitaline: thus, it had a bitter taste, and a disagreeable odor; its solution precipitated *tannic acid*, and responded to the acid and other tests (*vide* p. 492). The suspicious circumstances connected with the prisoner were of a very damaging character. It was shown that he had in his possession a large number of deadly poisons, among them digitaline; that he had at different times purchased this poison, to the amount of fifty-two grains, of which much had been used; and that this was altogether inconsistent with his requirements as a homœopathic practitioner. In short, he had the *motive*, the *opportunity*, and the *means* for accomplishing this purpose. The prisoner was condemned and executed. (See Tardieu and Roussin's treatise, p. 694, for full details of this interesting case.)

SECTION III.

POISONING BY COCCULUS INDICUS.

The Cocculus Indicus (*Levant nut*) is the fruit of the *Anamirta cocculus*, a tree growing in the East Indies. The kernel of the berry is the only poisonous part: it has an intensely bitter taste, and contains a highly poisonous principle, called *picrotoxine.* Cocculus Indicus is employed chiefly as a fish-poison, and also, in Great Britain, for the malicious destruction of game. It is also popularly believed to be extensively used for adulterating malt liquors, by imparting to them increased intoxicating properties with a diminished amount of hops and malt.

The *symptoms* produced by this drug are somewhat remarkable, and clearly indicate its action on the cerebro-spinal centres. Along with gastric irritation, there is a singular sort of narcotism, which is described by Dr. Taylor as "a strong disposition to sleep, and, at the same time, wakefulness. There is a heavy, lethargic stupor, with a consciousness of passing events, but a complete loss of voluntary power. It is a kind of nightmare feeling; altogether different from healthy sleep." (Prin. and Prac. of Med. Jurisp., 1873, p. 395.)

Only a few authenticated cases have been reported of poisoning with this substance, on man. One of these was a boy aged twelve years, who swallowed a composition for poisoning fish, containing Cocculus Indicus. It caused a burning pain in the gullet and stomach, not relieved by frequent vomiting. There was much febrile excitement, followed by delirium and purging, under which the patient sank on the nineteenth day. On inspection, the pia mater was congested with dark-colored liquid blood; there was serous effusion into the ventricles of the brain; the right lung was congested; and the abdomen presented all the marks of an advanced peritonitis. The coats of the stomach were discolored, and were softer and thinner than natural. (Canstatt, Jahresbericht, 1844–5, p. 298.)

The late Dr. Fish, of Philadelphia, has reported several cases of accidental poisoning by this substance, witnessed by himself while resident physician in the Philadelphia Hospital, Blockley. A strong decoction of the berries is used in that institution for the destruction of vermin upon the paupers. The vessel containing it was unfortunately placed near some tonic infusions in use by several patients. Through the ignorance of the nurse, a wineglassful of this decoction was given to each of three persons, and two tablespoonfuls to three others, in mistake for their usual medicine. Two of those who took the largest quantity were seized with convulsions about twenty minutes after taking the poison, and died in about half an hour. The contraction of the muscles was still apparent over twelve hours after death. The remaining four, who were seized within a few moments of each other, and within half an hour after swallowing the poison, exhibited faintness, mental confusion, giddiness, dimness of vision, nausea, excessive thirst, severe pain in the abdomen, and, in one case, insensibility. The pulse was much weakened, and the respiration was slow and labored. These all recovered under the use of emetics, and afterwards of mucilaginous drinks and stimulants; but they suffered greatly from headache during the rest of the day. (Wharton and Stillé, Med. Jurisp., 1873, ii. p. 596.) No post-mortem examination is stated to have been made in the above fatal cases.

The *external* application of this substance has been followed by violent and even fatal effects. Dr. W. B. Thompson, senior house-surgeon in the Emigrants' Hospital, New York, relates two instances of this character. A child aged six years, whose head, after the removal of the hair, had been washed with an alcoholic tincture of Cocculus Indicus, was seized, in less than half an hour after the application, with tetanic spasms. The pupils, during the convulsions, were extremely contracted, but in the interval between the spasms they were widely dilated. The child, although energetically treated, died in a few hours. On post-mortem examination, no changes of any kind were observed. A younger sister of the deceased, who had been subjected to the same process, was also attacked in a similar manner. Under the use of counter-irritation by mustard, and ingestions of the tincture of assafetida, she recovered, the convulsions gradually subsiding. The next day a scarlatinous eruption appeared upon the body and arms, which gradually faded during the day. (Phila. Med. Examiner, April, 1852.)

PICROTOXINE (*Picrotoxia*).—The active principle of Cocculus Indicus is generally considered to be an alkaloid, although in some of its reactions it differs from that class of bodies. It exists in the kernel of the berry in about the proportion of one per cent. It crystallizes in colorless, slender, six-sided prisms, having a silky gloss. It is sparingly soluble in cold water; more soluble in boiling water. It is very soluble in alcohol, ether, chloroform, and in amylic alcohol. Heated in a tube, it evolves an acid vapor like digitaline. Cold sulphuric acid does not affect it; but when warmed, it imparts to it an orange-yellow color, which becomes pale yellow by dilution, and brown if heated.

On the addition of bichromate of potassa, the green oxide of chromium is developed. Strong hydrochloric and nitric acids dissolve it without change of color. Neither tannic acid nor chloriodide of potassium and mercury precipitates it from its solutions. When boiled with a solution of potassa and the sulphate of copper, it precipitates the red oxide of copper, like grape-sugar. It is said to belong to the glucosides, like salicine (Taylor, *loc. cit.*, p. 396).

Unlike the alkaloids generally, it does not appear to combine with acids to form salts, but unites readily with bases: hence a weak solution of potash will dissolve it without difficulty. It may therefore be easily separated from the alkaloids by strongly acidulating the mixture and shaking it up with ether, which readily takes up the picrotoxine, and leaves the alkaloidal salts. From organic liquids, such as beer and porter, picrotoxine may be readily obtained by first acidulating with hydrochloric acid, and then shaking with ether, which holds the poison in solution and deposits it in crystals. This method has been successfully practiced by Mr. Langley (Pharm. Jour., Dec., 1862, p. 277). He was enabled to detect so small a quantity as one seven-hundred-and-fiftieth of a grain of picrotoxine in a pint of ale. He likewise separated it, by the same process, from the stomach of a cat that had been killed by this poison.

A curious case of attempted criminal poisoning by the unbroken berry of the Cocculus Indicus occurred in England (Reg. *v.* Clauderay) in 1849. Two berries had been given to an infant: one of these was vomited, and the other passed through the bowels unbroken; no bad effects followed. The accused was tried for poisoning, but urged in his defense that the *unbroken* pod did not come within the statute, not being properly "a poison or other destructive thing." The objection was overruled, and a verdict of guilty was rendered. On appeal, the judgment was sustained, and the prisoner was condemned. Here, the *intention* of the accused had undoubtedly great weight with the court.

There are several other vegetable poisons of minor importance: among them may be mentioned the bark and seeds of the *Laburnum* (*Cytisus Laburnum*), a very common tree or shrub of Great Britain. It contains an active poisonous principle, *cytisine*. Its effects are those of a narcotico-irritant. Both the bark and the seeds have produced fatal effects.

The leaves and berries of the *Yew* (*Taxus baccata*) act powerfully as an acrid, irritant narcotic, even in small quantities. Its active principle has not yet been isolated. Under the same head may be mentioned the berries of the *Privet* (*Ligustrum vulgare*); those of the *Guelder Rose* (*Viburnum opulus*); and those of the *Holly* (*Ilex aquifolium*).

INDEX.

A.

Absorption, action of poisons through, 22.
    circumstances influencing, 23.
    effect of, in removing poisons beyond the reach of analysis, 71.
    extreme rapidity of, 26.
Acetate of lead, 311.
    of morphia, 373.
Acetic acid, 174.
Aceto-arsenite of copper, 292.
Acid, acetic, 174.
    arsenic, 251.
    arsenious, 216.
    citric, 174.
    hydrochloric, 158.
    hydrocyanic, 474.
    igasuric, 396.
    meconic, 376.
    nitric, 149.
    oxalic, 162.
    phosphoric, 207.
    strychnic, 396.
    sulphuric, 137.
    tartaric, 173.
Acids, mineral, nature and effects of, 136.
Aconitia, properties of, 469.
    fallacies of tests for, 470.
    physiological test for, 470.
    separation from organic mixtures and the tissues, 470.
Aconite root, poisoning by, 465.
    fatal dose, 466.
    fatal period, 467.
    post-mortem appearances, 466.
    symptoms produced by, 465.
    treatment, 467.
Aconitum napellus, 465.
Alkalies, distinguishing properties of, 175.
    fatal quantity, 176.
    period when fatal, 176.
Alkalies, post-mortem signs of, 177.
    symptoms caused by, 175.
    treatment of poisoning by, 177.
Alkaloids, recovery by dialysis, 113.
        by Stas' method, 110.
        by Uslar and Erdmann's method, 383.
Aloes, poisoning by, 333.
Ammonia, 182.
    effects of vapor of, 176.
    period when fatal, 176.
    properties of, 182.
    salts of, 182.
    separation from organic mixtures, 183.
    symptoms produced by, 176.
Anæsthetics, 389.
Analysis, chemical, 67.
        accuracy of, 75.
        causes of failure in, 70.
        importance of, 68.
        objects of, 73.
        precautions to be observed in, 74.
        when impossible, 67.
Aniline, a source of fallacy, 422.
Antagonism of poisons, 91, 94.
Antimonetted hydrogen, 262.
Antimony, poisoning by, 253.
    necessity for extracting the metal, 268.
    poisonous salts of, 253.
    quantitative determination of, 269. (See *Tartar Emetic.*)
    recovery from the tissues, 270.
    separation from organic mixtures and the stomach, 265.
Appearances, post-mortem, liability of, to be confounded with the effects of diseases, 59.
    post-mortem, value of, in poisoning, 56.
Aqua ammonia, properties of, 182.
        poisoning by, 176.

Aqua fortis, 149.
Arsenic, metallic, 215.
    compounds of, 215.
  eaters of, 35, 215.
  white, 216.
Arsenious acid, 216.
  antidotes for, 226.
  antiseptic properties of, 224.
  detection after long periods, 225.
    in the stomach, 243.
    in the tissues, 245.
    in the urine, 250.
    in vomited matters, 242.
  external application of, 219.
  failure to detect, 222.
  fallacies in Reinsch's test, 239.
    in sulphuretted hydrogen test, 232.
  fatal dose, 220.
  fatal period, 221.
  liquid tests, 230.
  Marsh's test, 232.
    delicacy of, 238.
    fallacies in, 235, 236.
    modifications of, 236.
      Bloxam's, 241.
    nitrate of silver modification of, 237.
  post-mortem signs, 223.
  quantitative analysis of, 248.
  reduction process, 228.
  Reinsch's test for, 238.
    interferences in, 241.
  solubilities of, 216.
  sublimation test for, 227.
  sulphuretted hydrogen test, 231.
      interferences, 232.
  symptoms produced by, 216.
  taste of, 216.
  time of first manifestation of symptoms, 219.
  treatment for, 226.
  varieties of, 216.
Arsenite of potassa, 251.
Asagræa officinalis, 339.
Asthenics, 474.
Atropa belladonna, 436.
Atropia, 438.
  antagonism of, with morphia, 95.
  chemical properties of, 441.
  external application of, 439.
  physiological test, 443.
  poisoning by, 438.
  post-mortem signs, 441.
  recovery from the blood, 443.
  separation from complex organic mixtures, 442.
  subcutaneous injection of, 439.
  tests for, 441.
  treatment of poisoning by, 441.

B.

Belladonna, poisoning by, 436.
  poisoning by, treatment for, 441.
  symptoms produced by, 436.
Bichromate of potassa, 330.
Binoxalate of potassa, 172.
    poisonous symptoms, 172.
Bismuth, subnitrate of, 327.
  arsenic in, 328.
Bittersweet, 449.
Bloxam's method for detecting arsenic, 241.
Blue vitriol, 293.
Bromine, poisoning by, 213.
Brucia, poisoning by, 434.
    physiological effects of, 434.
    tests for, 435.
  solubility, 434.
  special chemical properties, 435.
  test for nitric acid, 153.
Burnett's disinfecting fluid, 323.

C.

Calabar bean, poisoning by, 471.
  effect on system, 472.
  chemical properties of physostigmia, 473.
Carbolic acid, poisoning by, 342.
  chemical analysis, 343.
  fatal quantity, 343.
  post-mortem appearances, 343.
  symptoms, 342.
  treatment, 344.
Case of Buffenbarger, 41.
  of Castaing, 68, 372.
  of the Count Bocarmé, 49, 87.
  of De la Pommerais, 460.
  of Donellan, 48, 68.
  of Donnall, 77, 231.
  of the Duke of Praslin, 218.
  of Hartley, 88.
  of Hendrickson, 470.
  of Mr. Hodgson, 89.
  of Humphrey, 88.
  of Mrs. E. E. Lloyd, 65, 328.
  of North, 89.
  of Palmer, 403, 433.
  of M. Pralet, 49.
  of Dr. Paul Schoeppe, 93, 485.
  of Sprague, 86.
  of Madeline Smith, 74.
  of Mrs. E. G. Wharton, 50, 267.
Castor-oil beans, poisoning by, 334.
Causes modifying the effects of poisons, 33.
Cerebral neurotics, 361.
Cerebro-spinal neurotics, 436.

Chemical analysis, failure of, 70.
    importance of, 67.
    objects of, 73.
  decomposition of poisons, 72.
  reagents, purity of, 80.
  tests, fallacies in, 79.
Chloride of zinc, poisoning by, 323.
Chlorinated soda, poisoning by, 188.
Chromium, poisoning by, 330.
Classification of poisons, 132.
Cocculus Indicus, poisoning by, 496.
Codeia, physiological effects of, 379.
  properties of, 379.
  tests for, 379.
Colchicina, properties of, 335.
Colchicum, poisoning by, 334.
    morbid appearances, 335.
Color tests, fallacy of, 77.
Compensation of medical experts, 123.
Compound poisoning, 91.
Conia, distinguished from nicotina, 461.
  effects of, 454.
  general properties of, 454.
  separation from the blood and tissues, 455.
  solubility, 454.
Conium maculatum, 452.
Copper, metallic, 289.
    poisoning by, 289.
  contamination of food by, 289.
  effects of salts of, on the system, 292.
  fatal dose of sulphate of, 294.
  fatal period, 294.
  post-mortem signs, 295.
  quantitative determination, 304.
  recovery from organic mixtures, 299.
    from the stomach and tissues, 301.
    from the urine, 303.
  subacetate of, 296.
  sulphate of, 295.
  symptoms, 293.
  tests, 296.
  treatment of poisoning by, 294.
Corrosive sublimate, poisoning by, 273.
    symptoms, 274.
    tests for, 278.
    treatment, 277.
  chemical properties of, 274.
  chronic poisoning by, 277.
  external application, 276.
  failure to detect, 284.
  fatal dose, 276.
  fatal period, 276.
  post-mortem appearances, 277.
  quantitative analysis, 288.
Corrosive sublimate, recovery from organic mixtures, 282.
    from the tissues, 283.
    from the urine, 283
  reduction test, 280.
  salivation from, 278, 285.
Croton oil, poisoning by, 332.
Curara, 422.
Curarine, 422.

D.

Danger and Flandin's method for arsenic, and other metals, 100, 249.
Datura stramonium, 444.
Daturia, chemical properties of, 446.
  separation from organic mixtures, 447.
  tests for, 446.
Deliriants, or delirifacients, 436.
Depressants, 452.
Dialysis, method of, 113.
Digitaline, 491.
  chemical reactions of, 492.
  detection in organic mixtures, 493.
    case of De la Pommerais, 495.
Digitalis, poisoning by, 490.
    post-mortem appearances, 491.
    symptoms of, 490.
Disease, effects of, confounded with those of poisons, 58.
  modifying influence of, on the action of poisons, 36.
Diseases simulating poisoning, 52.
Duties and privileges of medical experts, 119.

E.

Elaterium, poisoning by, 333.
Elimination of poisons, 27.
Evidence from chemical analysis, 67.
  from circumstances, 87.
  from experiments on animals, 80.
  from morbid lesions, 56.
  from symptoms, 45.
Evidences of poisoning, 45.
Experts, medical, rights of, 123.

F.

Failure to detect poisons, 70.
Fresenius and Babo's method for detection of metallic poisons, 99, 246.

H.

Habit, modifying influence of, on poison, 35.
Hellebore, American, 337.
  black, 337.

Hellebore, white, poisoning by, 338.
        symptoms of, 337.
        treatment, 338.
Hemlock, poisoning by, 452.
        post-mortem appearances, 453.
        symptoms, 453.
        treatment, 454.
    properties of, 452.
    water, 456.
Hydrochloric acid, 158.
    fatal dose, 158.
    fatal period, 158.
    post-mortem signs, 158.
    properties of, 158.
    quantitative analysis, 162.
    separation from organic mixtures, 160.
    symptoms, 158.
    tests for, 160.
    treatment of poisoning by, 158.
Hydrocyanic acid, 474.
    external application of, 477.
    fatal dose, 477.
    fatal period, 476.
    post-mortem appearances, 478.
    properties of, 474.
    quantitative determination, 487.
    separation from organic mixtures, 484.
        from the tissues and blood, 484.
    symptoms produced by, 475.
    tests for, 479.
    treatment of poisoning by, 478.
Hyoscyamia, 448.
Hyoscyamus, poisoning by, 447.
        chemical analysis, 448.
    fatal dose of, 447.

I.

Idiosyncrasy, influence of, 36.
Igasuric acid, 396.
Imbibition, post-mortem, of poisons, 38.
Indian poke, 337.
Intestines, perforation of, 61.
Iodine, poisoning by, 209
Irritant poisons, effects of, 135.
    poisoning, morbid appearances in, 136.

J.

Jamestown weed, poisoning by (see *Stramonium*), 442.

L.

Laudanum, 362.
Lead colic, 313.
    poisoning by, 305.
Lead, accidental poisoning by, 306.
    acetate of, effects of, 311.
        chronic poisoning by, 313.
        fatal dose, 312.
        fatal period, 312.
        post-mortem signs, 313.
        properties of, 311.
        quantitative analysis, 321.
        symptoms of poisoning by, 311.
        treatment of poisoning by, 312.
    contamination of water by, 309.
    detection in the urine, 321.
    external application of, 308
    separation from organic mixtures, 318.
    tests, 316.
Lobelia, poisoning by, 463.

M.

Marsh's test for antimony, 262.
    test for arsenic, 232.
Meconic acid, 376.
    chemical properties of, 377.
    fallacies in detecting, 377.
    iron test for, 377.
    physiological effects of, 377.
    separation from organic mixtures, 381.
    tests for, 377.
Meconine, 380.
Medico-legal questions, 114.
Mercury, metallic, 272.
    compounds of, 273, 288.
    detection in the organs, 283.
        in the urine, 283.
    physiological effects of, 273.
    protochloride (calomel), 288.
Metal, necessity of obtaining the, 76.
Method of chemical procedure in the search for poisons, 98.
Microscope, application of, 66.
Mineral acids, 137.
Monkshood, 465.
Morphia, 371.
    effects of, 371.
    external application of, 374.
    failure to detect, 380.
    fatal dose, 373.
    fatal period, 366.
    general chemical properties, 375.
    post-mortem appearances, 374.
    recovery from stomach, 380.
        from the blood, 383.
        from the tissues, 383.
        from the urine, 384.
    separation from organic mixtures, 380.

Morphia, symptoms of poisoning by, 371.
  tests for, 374.
  treatment of poisoning by, 370.
Muriatic acid, 158.

N.

Narceia, or narceine, physiological action of, 379.
  properties of, 379.
  tests for, 379.
Narcotico-irritant poisoning, 132.
Narcotics, 361.
Narcotina, physiological action of, 378.
  properties of, 378.
  test for nitric acid, 153, 156.
  tests for, 378.
Neurotics, properties of, 133.
Nicotia, or nicotina, 459.
  fatal dose of, 459.
  fatal period, 459.
  post-mortem signs, 458.
  recovery from tissues and blood, 462.
  symptoms produced by, 457.
    case of the Count Bocarmé, 460.
Nicotiana tabacum, properties of, 457.
Nightshade, deadly (belladonna), 436.
  garden, 449.
  woody, 449.
Nitrate of potassa, 184.
Nitric acid, poisoning by, 149.
  antidotes for, 151.
  fatal dose, 150.
  fatal period, 150.
  fumes of, fatal, 150.
  general chemical properties, 152.
  post-mortem appearances in poisoning by, 151.
  recovery from organic substances, 154.
  stains produced by, 150, 157.
  symptoms produced by, 149.
  test for, 152.
Nux vomica, properties, 396.
  fatal dose, 396.
  symptoms produced by (see *Strychnia*).
  treatment of poisoning by (see *Strychnia*).
  uses, 397.

O.

Œsophagus, tying of, 83.
Oil of vitriol, 137.
Opianyl, physiological effects of, 380.
  properties of, 380.
  tests, 380.
Opium, nature and properties of, 362.
  external application of, 368.
  failure to detect, 374.
  fatal dose, 366.
  fatal period, 365.
  inequality of strength of, 362.
  post-mortem appearances, 369.
  recovery from organic mixtures, 380.
  separation from stomach and tissues, 380.
  symptoms produced by, 363.
  tests for, 374.
  time of symptoms, 365.
  treatment of poisoning by, 370.
Orpiment, 253.
Oxalic acid, properties, 162.
  fatal dose, 164.
  fatal period, 164.
  post-mortem lesions, 165.
  recovery from organic mixtures, 167.
    from the tissues and urine, 171.
  stains produced by, 171.
  symptoms produced by, 163.
  treatment of poisoning by, 165.

P.

Papaver somniferum, 362.
Phosphoric acid, 207.
    tests for, 267.
Phosphorus, 194.
  fatal dose of, 195.
  fatal period, 196.
  fatty degeneration occasioned by, 198.
  hydrogen test, 205.
  Lipowitz's test, 204.
  Mitscherlich's test, 203.
  post-mortem signs, 197.
  properties of, 201.
  quantitative determination, 206.
  recovery in the free state, 202.
    from organic mixtures, 202.
  symptoms, acute, 194.
    chronic, 195.
  treatment of poisoning by, 196.
  varieties of, 208.
Physostigma venenosum, 471.
Physostigmia, 473.
Picrotoxia, 498.
Poison, definition of a, 14.
Poisoned flesh, 360.
Poisonous cheese, 355.
  fish and mussels, 356.
  meats, 358.
  sausages, 353.
Poisons, classification of, 132.
  failure to detect, 70.

Polarized light a test for soda, 181.
Post-mortem appearances as evidences of poisoning, 56.
  examinations, proper mode of conducting, 62.
  imbibition of poisons, 38.
Potassa, poisoning by, 175.
  fatal quantity, 176.
  fatal period, 176.
  post-mortem appearances, 177.
  quantitative determination, 181.
  recovery from organic mixtures, 180.
  salts of, poisoning by, 184.
  special chemical properties, 178.
  symptoms, 175.
  tests for, 179.
  treatment of poisoning by, 177.
Prussic acid (see *Hydrocyanic Acid*).

R.

Ratsbane. 215.
Reagents, chemical, impurities in, 80.
Realgar, 253.
Reinsch's test, 238, 270.

S.

Sabadilla, 339.
Soda, poisoning by, 175.
  properties of, 181.
  recovery from organic mixtures, 182.
  tests for, 181.
Solania, properties of, 450.
  symptoms produced by, 449.
Solanum dulcamara, 449.
    symptoms produced by, 449.
  nigrum, 449.
  tuberosum, 450.
Spinal neurotics, or tetanics, 396.
Spirit of salt, 158.
Stas' process for recovering the alkaloids, 110.
St. Ignatius's bean, 396.
Stomach, redness of, 59.
  rupture of, 56.
  softening of, 61.
  ulceration and perforation of, 60, 61.
Stramonium, poisoning by, 442.
  effects on the system, 444.
  post-mortem signs, 446.
  properties of, 442.
  seeds, poisoning by, 442.
  treatment of poisoning by, 446.
Strychnia, poisoning by, 396.
  bitterness of taste, 413.
Strychnia, color test for, 414.
    delicacy of, 416.
    effect of morphia on, 418.
    fallacies of, 422.
    interferences with, 417.
  external application of, 401.
  failure to detect, 433.
  fatal dose, 401.
  fatal period, 403.
  frog test for, 426.
  galvanic test, 423.
    period of invasion of symptoms, 399.
    physiological test, 426.
  poisoning, diagnosis of, 408.
    post-mortem appearances, 407.
  properties of, 397.
  recovery from organic mixtures, 428.
    from the stomach, 428.
    from the tissues and blood, 431.
    from the urine, 433.
  tests for, 423.
  treatment of poisoning by, 404.
Strychnos nux vomica, 396.
  Ignatia, 396.
Sugar of lead, poisoning by, 311.
Sulphate of copper, 292.
  of indigo, 154.
  of lead, 316.
  of zinc, 322.
Sulphuric acid, absorption and elimination of, 142.
  fatal dose, 140.
  fatal period, 139.
  general chemical properties, 142.
  poisoning by, 137.
    post-mortem appearances, 140.
  properties of, 137.
  quantitative determination, 148.
  recovery from organic mixtures, 144.
    from the stomach, 145.
  stains produced by, 147.
  symptoms produced by, 137.
  tests for, 142.
  treatment of poisoning by, 140.
Symptoms, as evidences of poisoning, 45.

T.

Tartaric acid, poisoning by, 173.
Tartar emetic, poisoning by, 254.
  chemical properties of, 254.
  external application, 260.
  fatal dose, 256.
  fatal period, 257.

Tartar emetic, importance of obtaining the metal, 268.
  post-mortem signs, 258.
  quantitative analysis, 269.
  recovery from organic mixtures, 265.
    from the stomach, 266.
    from the tissues and blood, 270.
    from the urine, 272.
  symptoms produced by, 254.
  tests for, 260.
  treatment of poisoning by, 260.
Tetanics, 396.
Tetanus, distinguished from strychnia-poisoning, 55.
Thorn-apple, 444.
Tin, poisoning by, 329.
Tincture of opium, 362.
Tobacco, poisoning by, 457.
    symptoms of, 457.
  external application of, 457.
  smoking of, 458.
Trichiniasis, 353.
  symptoms of, 354.

U.

Uslar and Erdmann's method of recovering the alkaloids, 383.

V.

Vegetable irritants, 331.
Veratria, poisoning by, 339.
  chemical properties of, 339.
  recovery from organic mixtures, 341.
  symptoms, 340.
  test for sulphuric acid, 144.
  tests, 340.
Veratrum album, poisoning by, 338.
    symptoms, 338.
  viride, poisoning by, 337.
    symptoms, 337.
Verdigris, 296.

W.

White hellebore, 338.
  vitriol, 322.
Wolfsbane, 465.
Woorara, properties of, 422.

Z.

Zinc, poisoning by, 322.
  post-mortem appearances, 323.
  recovery from organic mixtures, 325.
  sulphate, 322.
  symptoms of poisoning by, 322.
  tests, 325.
  treatment of poisoning by, 324.

THE END.

5-

UC SOUTHERN REGIONAL LIBRARY FACILITY

A 000 552 467 3